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Correlation Among Optic Nerve Head Changes, Visual Field Changes and Retinal Nerve Fiber Layer Thickness in Primary Open Angle Glaucoma

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Original Research Article

ABSTRACT

A prospective cross-sectional non interventional type of study was conducted among cases of primary open glaucoma (POAG).The purpose of the study was to understand correlation among optic nerve head changes, visual field changes and retinal nerve fiber layer thickness in cases of primary open angle glaucoma. This study, conducted among 72 patients with 138 eyes, established a positive correlation between mean vertical CDR (r 0.5726) as compared with mean RNFL thickness and a negative and weak positive correlation between mean vertical CDR compared to visual field parameter mean defect MD (r -0.2364) and PSD (r 0.3516) respectively. The p value between CDR and RNFL thickness was 0.00001, which was statistically significant.

KEY WORDS: optic nerve head (ONH), primary open angle glaucoma (POAG), retinal nerve fiber layer (RNFL), visual field (VF)

INTRODUCTION:

Primary open angle glaucoma (POAG) is the most common type of glaucoma. POAG can be considered chronic progressive optic neuropathy which is accompanied by a characteristic cupping and atrophy of the optic disc, visual field (VF) loss, open angles and no obvious ocular or systemic reason. The disease is characterized by progressive loss of retinal ganglion cells and their axons associated with tissue remodelling in the optic nerve head (ONH). The concepts and definitions of glaucoma have evolved in the past 100 years[1]and still they remain imprecise and subject to technical qualifications. Glaucoma is a leading cause of irreversible blindness throughout the world. World Health Organization statistics, published in 1995, indicate that glaucoma accounts for blindness in 5.1 million persons, or 13.5% of global blindness (behind only cataracts and trachoma at 15.8 million persons, or 41.8% of global blindness, and 5.9 million, or 15.5%, respectively) [2]. Worldwide, it has become the second most common cause of bilateral blindness. Open-angle glaucoma and angle-closure glaucoma were estimated to affect approximately 66.8 million persons by the year 2000, with 6.7 million experiencing bilateral blindness[3]. The word glaucoma originally meant 'clouded' in Greek; as such, it may have referred either to a mature cataract or to corneal edema that might result from chronic elevated pressure. Today the term does not refer to a single disease entity, but rather to a group of diseases that differ in their clinical presentation, pathophysiology, and treatment. These diseases are grouped together because they share certain features, including cupping and atrophy of the optic nerve head, which has attendant visual field loss and is frequently related to the level of intraocular pressure (IOP).

Whether manifesting as POAG, primary angle-closure, or congenital disease, glaucoma is the second leading cause of blindness worldwide, with a disproportional morbidity among women and Asians [4-7]. The major mechanism of visual loss in glaucoma is retinal ganglion cell apoptosis, leading to thinning of the inner nuclear and nerve fiber layers of the retina and axonal loss in the optic nerve. The optic disc becomes atrophic, with enlargement of the optic cup. Diagnosing POAG requires evaluation of IOP, the anterior chamber angle (by gonioscopy), optic disc, and visual field. In glaucoma, there may be concentric enlargement of the optic cup or preferential superior and inferior cupping with focal notching of the rim of the optic disc. As cupping develops, the retinal vessels on the disc are displaced nasally (Figure1). Typical

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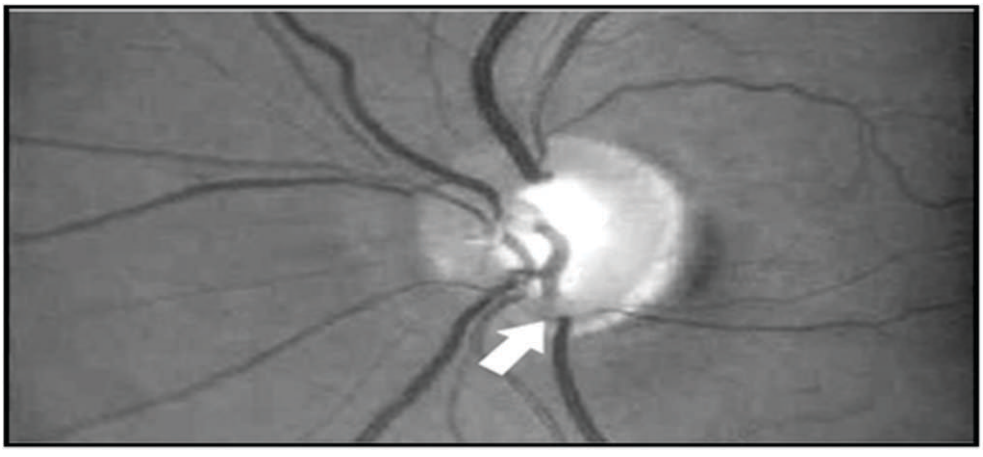


Figure 1: Early glaucoma showing inferior focal notching of the neuroretinal rim (arrow).

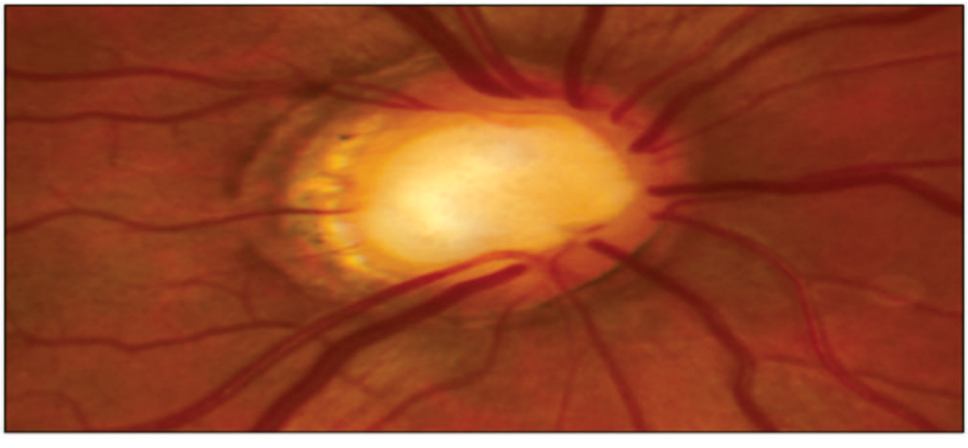


Figure 2: Typical glaucomatous cupping in right eye.

glaucomatous cupping in right eye (Figure 2).

Glaucomatous field loss involves mainly the central 30 degrees of field (Figure 2,3). The earliest change is baring of the blind spot. Contiguous extension into Bjerrum's area of the visual field at 15 degrees from fixation-produces a Bjerrum scotoma and then an arcuate scotoma. Focal areas of more pronounced loss within Bjerrum's area are known as Seidel scotomas. Double arcuate scotomas above and below the horizontal meridian are often accompanied by a nasal step (of Roenne) because of differences in size of the two arcuate defects. Peripheral field loss tends to start in the nasal periphery as a constriction of the isopters. Subsequently, there may be connection to an arcuate defect, producing peripheral breakthrough. The temporal peripheral field and the central 5-10 degrees are affected late in the disease.

***Optical Coherence Tomography (OCT) findings in POAG*:**

Optic Nerve Head Changes: The *optic nerve head,* or *optic disc,* is usually round or slightly oval in shape and contains a central *cup.* The tissue between the cup and the disc margin is called the *neural rim* or *neuroretinal rim.* In normal individuals, the rim has a relatively uniform width and a color that ranges from orange to pink.

Retinal Nerve Fiber Layer (RNFL) thickness: RNFL thinning is a sensitive indicator of the extent of glaucomatous damage and that RNFL loss precedes measurable ONH and VF damage approximately six years before any detectable VF defects. Thus, the possibility of detecting these defects in areas of physiological decreased visibility is enhanced, when OCT is used. Accurate and objective methods of detecting disc and RNFL abnormalities, and their progression, would facilitate the diagnosis and monitoring of glaucomatous optic neuropathy.

MATERIALS & METHODS:

Total 72 patients (6 patients, uniocular) were included in this study which was conducted in

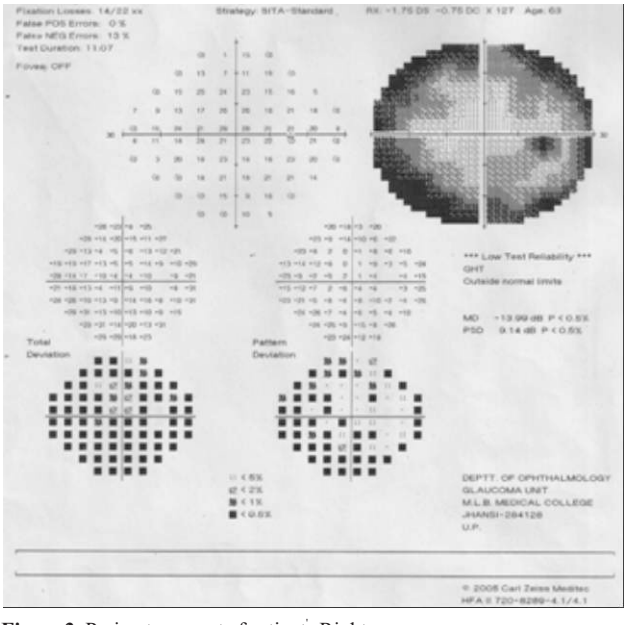


Figure 3: Perimetry report of patient's Right eye.

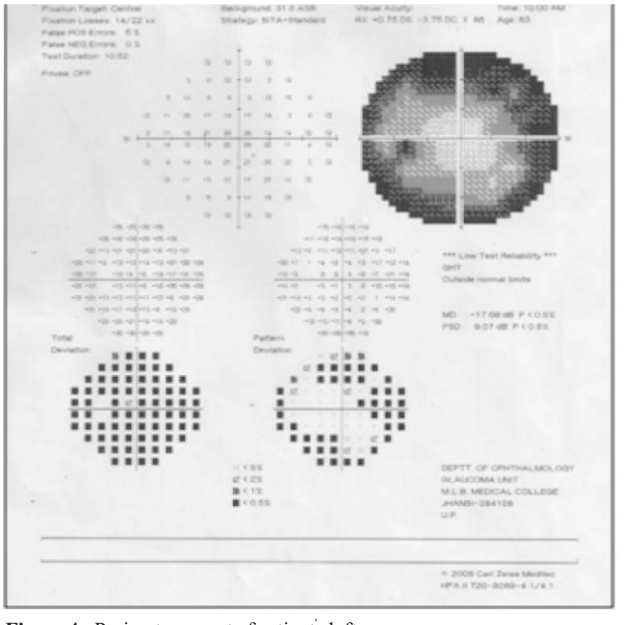


Figure 4: Perimetry report of patient's left eye.

Department of Ophthalmology, MLB medical college, Jhansi, India over a period of 18 months (i.e. April 2018 to September 2019). All procedures were followed according to Helsinki Declaration of 1975, as revised in 2000. The necessary permission from the Ethical and Research Committee was obtained for the study.

The POAG patients were included if the following inclusion criteria was satisfied: (a) Age ≥40 years; (b) Known/ newly diagnosed patient with unilateral or bilateral primary open angle glaucoma with either (i) Visual acuity or Best corrected visual acuity 6/24; or (ii) Elevated intraocular pressure (IOP) (greater than 21mm Hg) on at least two separate visits; or (iii) Glaucomatous optic disc appearance; (c) Wide and open angle on gonioscopy with goldmann gonio lens[Grade 3 and above by Shaffer, classification]; and (d) Those who were physically fit for perimetry and OCT evaluation.

The criteria for exclusion were: (a) Age < 40years; (b) Patients with diabetes, hypertension or any other chronic disorders like uveitis, retinal detachment or retinal vascular disorders; (c) Other forms of glaucoma like acute angle closure glaucoma, steroid induced glaucoma etc (all forms of secondary glaucoma); (d) History of ocular trauma and Recent ocular surgery (<6 months); (e) Patients with very high refractive error more than [± 5.0 D]; (f) Pregnancy or Lactation.

All the patients reported in Out Patient Department of Ophthalmology were evaluated, as per inclusion and exclusion criteria of the study, for diagnosis of primary open angle glaucoma. The status of each patient was evaluated by Slit lamp examination, Visual Acuity, Best Corrected Visual Acuity (BCVA), Intra Ocular Pressure by Non Contact Tonometer, Fundus examination (clinical vertical CDR), OCT(VCDR and RNFL Thickness (Retinal nerve fiber layer Thickness),and Perimetry (Visual field parameters i.e. Mean Deviation (MD), Pattern Standard Deviation (PSD). Quantitative data as mean and qualitative variables were expressed using percentage. The Student's paired 't" test for equal or unequal variances was used. The p-value of < 0.05 for one - tailed hypothesis was considered statistically significant to reject the 'null hypothesis', if the t test value or the observed difference between two means is greater than 2 times of standard error of difference (SED), the 5% level of significance. All statistical calculation/descriptive analyses were made with the help of data analyses tool of Microsoft Excel 2016 as shown below: Calculation of total sum, mean/average & standard deviation (SD) of each data group. Obtaining p value between study parameter by student's paired t-test of two independent mean. Scatter diagram also made for showing correlation in VCDR and RNFL Thickness, VCDR and MD (Visual field parameter) VCDR and PSD (Visual field parameter). Calculation of correlation coefficient(r) of both eyes data by Pearson's Correlation Coefficient formula. The Pearson's coefficient (r) can take range of values from +1 to -1.

The patient's protocols were recorded in data collection form and all values were taken from raw

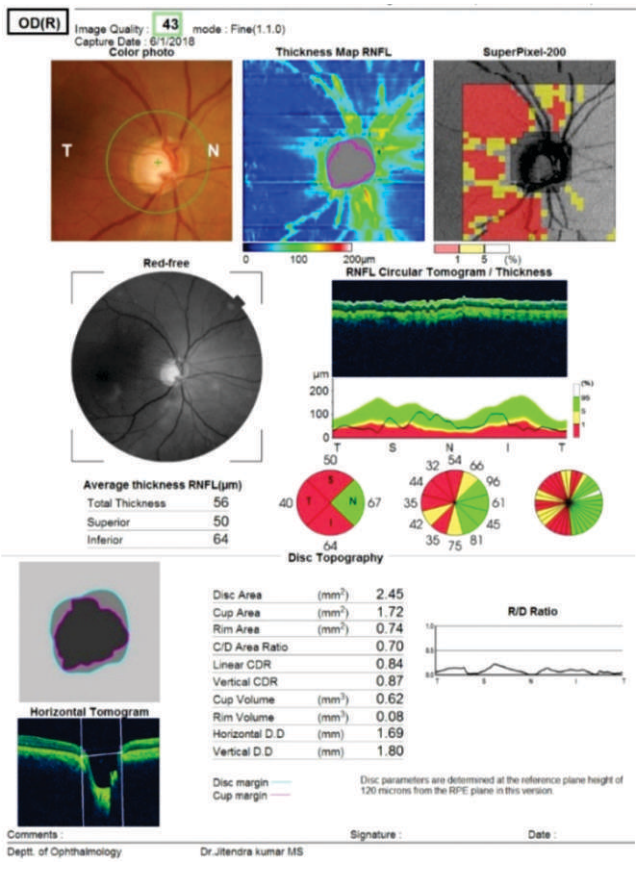


Figure 5. OCT –Inferior ,superiorand temporal thinning (right Eye).

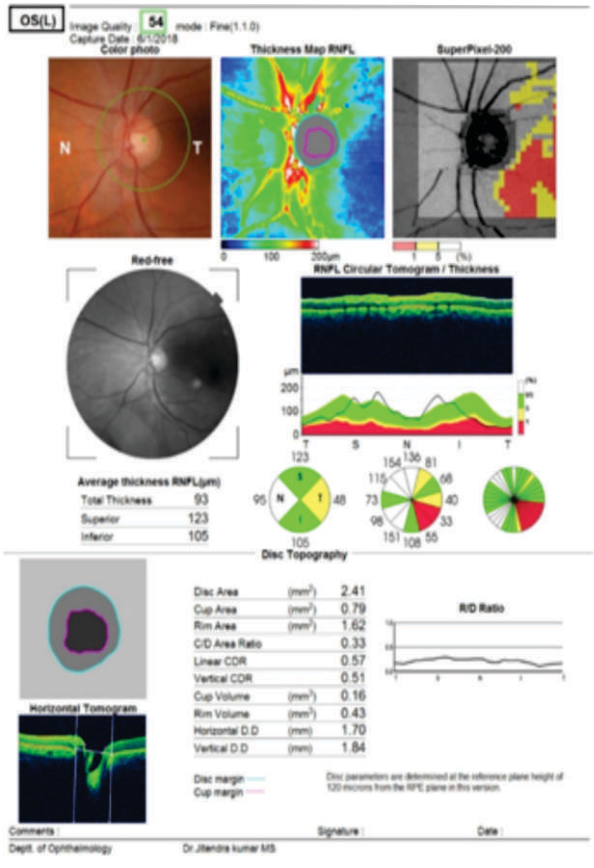


Figure 6.OCT - RNFL thinning in inferior(left eye)

data of the total patients.

RESULTS:

Seventy Two patients with primary open angle glaucoma fulfilled the inclusion and exclusion criteria of this study. The mean age in years was 59.44 ±11.64 (Range 42to 89 years) of total patients. The majority of POAG patients were age group above 50 year (73.6%). Sex wise distribution shows, 42 (58.33%) male and 30(41.67%) female. The male female ratio was 1.4:1.

The mean vertical CDR was 0.54 (±0.145) in right eye and 0.54 (±0.106) in left eye compared with Mean RNFL (µm) of 93.87(±24.71) in right eye and 93.35(±24.12) in left eye of all the patients. The "p value" was 0.00001. indicating that there was statistically significant difference in VCDR and RFNL thickness changes of right eyes of patients. The Pearson's correlation coefficient value of right eye was 0.5726, suggestive of moderate positive correlation between VCDR and RNFL thickness changes in right eye.With the "p value" was 0.1169, indicating that there was no statistically significant difference in VCDR and RFNL thickness changes of left eyes of patients. The Pearson's correlation coefficient value of left eye was 0.1864, suggestive that weak positive correlation between VCDR and RNFL thickness changes in left eye.

The Mean Deviation (MD) of -4.29 (±2.767) in right eye and -4.40 (±02.807) in left eye of all the patients. The 'p value' was 0.025382, indicating that there was statistically significant difference in CDR and visual field parameter Mean Deviation (MD) changes of right eyes of patients. The correlation coefficient (r) value of right eye was -0.2634, suggesting a weak negative correlation between CDR and MD in right eye. The 'p value' was 0.00093, indicating that there was statistically significant difference in CDR and MD changes of left eyes of patients. The correlation coefficient (r) value of left eye was -0.38, suggesting a weak negative correlation between CDR and MD changes in left eye.

The Pattern Standard Deviation (PSD) of +3.77(±3.05) in right eye and +3.29(±2.21) in left eye of all the patients. The "p value" was 0.0024, indicating that there was statistically significant difference in CDR and visual field parameter Pattern

Table 1: Age wise distribution of patients.

S. No	Age group (in years)	Total number of patients	Percentage (%)
1.	40-44	9	12.5
2.	45-49	10	13.9
3.	50-54	10	13.9
4.	55-59	8	11.1
5.	60-64	9	12.5
6.	65-69	11	15.3
7.	70 and above	15	20.8
Total	59.44 ± 11.64	72	100%

Table 2: Gender wise distribution of patients.

Gender	Male	Female	Total
No of patients	42	30	72
Percentage	58.33 %	41.67 %	100%

Table 3: Mean CDR (Cup: Disc ratio) of total patients on OCT.

CD (OCT)	Total patients		t test score	p value
	Right eye	Left eye		
Mean	0.54	0.54	0.2098	0.4170
SD	±0.145	±0.106		

Table 4: Mean Retinal Nerve Fiber (RNFL in µm) thickness of patients.

RNFL thickness (µm) in OCT	Total patients			
	Right eye	Left eye	t test score	p value
Mean	93.87	93.35	0.3624	0.3587
SD	±24.71	±24.12		

Standard Deviation (PSD) changes of right eyes of patients. The correlation coefficient (r) value of right eye was 0.3516, suggesting a weak positive correlation between CDR and MD in right eye. The "p value" was 0.7154, indicating that there was not statistically significant difference in CDR and visual field parameter Pattern Standard Deviation (PSD) changes of left eye of patients. The correlation coefficient (r) value of left eye was 0.0437, suggesting a very weak positive correlation between CDR and PSD in left eye.

DISCUSSION:

In our study, we have analyzed technique of quantifying disc changes. Vertical CDR (Optic nerve head changes) correlates better with RNFL thickness and visual field changes in Glaucoma patients. Glaucoma is major public health problem, a leading cause of irreversible blindness throughout the world and the second leading cause of world blindness. It accounts for 15% of global blindness. The regional burden of blindness (RBB) is highest for India (23.5% of global blindness) [8]. With such prevalence rates, it is imperative to find measures to detect the disease in early stages before it starts to cause visual morbidity. By 2020, the number of glaucoma patients is expected to be 76 million and by 2040 this number will increase to 112 million. [9,10,11]

Table 5:Visual field mean deviation (VFMD) in perimetry of patients.

	Total Patients		t test score	p value
Visual field test Parameter	Right eye	Left eye		
Mean Deviation (MD)	-4.29	-4.40	0.2248	0.4112
SD	±2.76	±2.80		

Table 6: Visual field parameter mean defect in both eye of total patients.

	Total patients		t test score	p value
Visual field test Parameter	Right eye	Left eye	1.085	0.1398
Pattern Standard Deviation (PSD)	+3.77	+3.09		
SD	±3.050	±2.217		

Another study [12] found that men were 1.37 times more likely to have open angle glaucoma than women, although gender wise prevalence of POAG has always been controversial. Majority of our subjects (55%) were from low socio-economic strata which resonates with the study conducted for assessment of the rates of blindness and of partial sight registration in glaucoma patients [13] inferring that low socio-economic background was indeed a risk factor for development of glaucoma despite universal health care. A study published in journal of Ophthalmology [14] aimed to compare SD-OCT evaluation of RNFL thickness in normal controls and POAG of various stages and found that normal patients had the thickest RNFL thickness when compared with patients. Moreover, increased glaucoma severity was associated with thinner RNFL. In a study conducted for correlation of average thickness using the SRRATUS OCT with the

Table 7: Correlation between VCDR and RNFL Thickness.

Total patients	VCDR (µm) in OCT (Mean ± SD)	RNFL (µm)	Correlation (r)	p value
Right eye	0.54 ± 0.145	93.87 ± 24.716	0.5726	0.00001
Left eye	0.54 ± 0.106	93.35 ± 24.125	0.1864	0.1169

Graph 1: Scatter diagram between VCDR changes and RNFL thickness changes

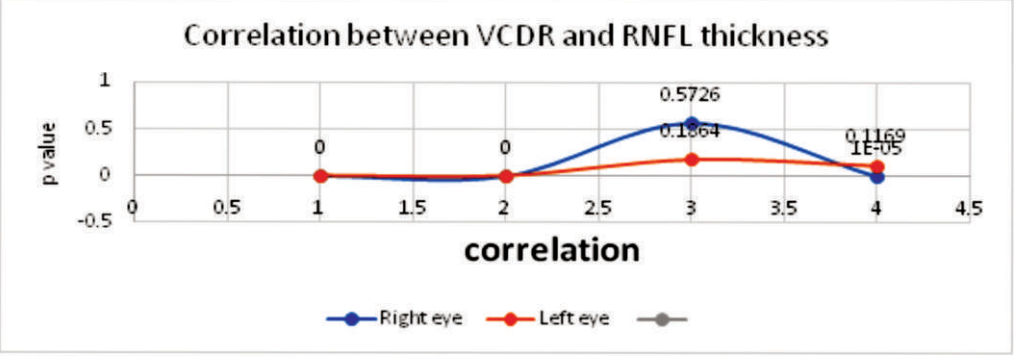
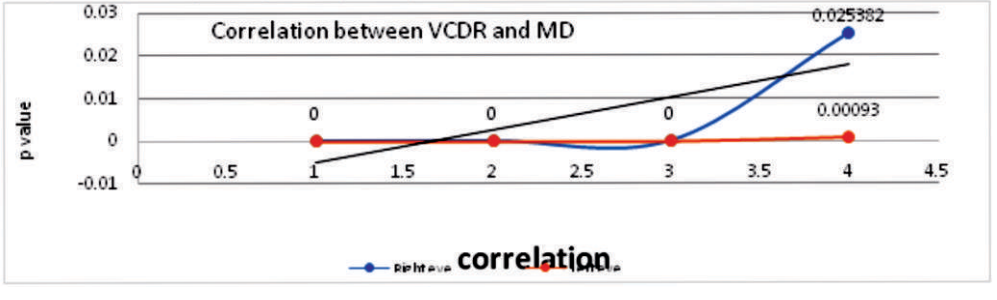


Table 8: Correlation between CDR and Mean Deviation (MD).

Total patients	CDR (µm) in OCT (Mean ± SD)	MD (dB) (Mean ± SD)	Correlation (r)	p value
Right eye	0.54 ± 0.145	-4.29 ± 2.767	-0.2634	0.025382
Left eye	0.54 ± 0.106	-4.40 ± 2.807	-0.38	0.00093

Graph 2: Scatter diagram between CDR changes and Visual field parameter (MD changes.



perimetric staging of glaucoma demonstrated that NRR area correlates more strongly with field damage than C/D ratio or cup volume [15]. However, the correlation between VCDR and RNFL thickness in the present study is more strongly than visual field parameter (MD, PSD) due to small sample size studied over short span of time. Since our study included less advanced glaucoma cases, it could be expected that it would not reveal such high correlation between RNFL thickness and visual field parameters (MD ,PSD).

In subjects with early glaucoma, evaluation of the RNFL is important for evaluating glaucomatous ganglion cell loss. Kanamori et al. [22] showed that the RNFL decreased in glaucomatous eyes, with or without early visual field defects. This study comprising total 72 patients with 138 eyes, established a positive correlation between mean vertical CDR (r 0.5726) compared with mean RNFL thickness and a negative and weak positive correlation between mean vertical CDR compared to visual field parameter mean defect MD (r -0.2364) and PSD (r 0.3516) respectively. The p value between CDR and RNFL thickness was 0.00001, which was statistically significant.

Each 1 mmHg rise in IOP during a median follow-up time of 5.3 years has been shown to be

Table 9: Correlation between CDR and PSD.

Total patients	CDR (µm) in OCT (Mean ± SD)	PSD (dB) (Mean ± SD)	Correlation (r)	p value
Right eye	0.54 ± 0.145	+3.77 ± 3.050	0.3516	0.0024
Left eye	0.54 ± 0.106	+3.29 ± 2.217	0.0437	0.7154

Graph 3: Scatter diagram between CDR (OCT) and visual field parameter (PSD).

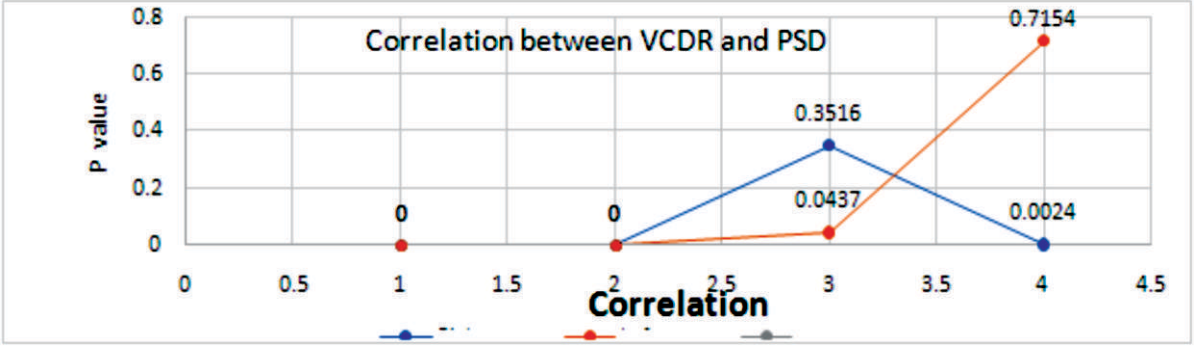


Table 10: Strength of correlation between different parameters.

Study variables	p value	Pearson's coefficient (r)
VCDR and RNFL Thickness	0.00001	0.5726
VCDR and MD	-0.2538	-02364
VCDR and PSD	0.0024	0.3516

Table 11: Summary of P values showing difference between correlation.

Study variables	p value	Pearson's correlation coefficient (r)	Statistical significance
VCDR and RNFL Thickness	0.00001	0.5726	Significance at 5% significance level
VCDR and MD	0.025382	-0.2364	Significance at 5% significance level
VCDR and PSD	0.0024	0.3516	Significance at 5% significance level

Table 12: Correlations between RNFL thickness and Visual field parameter (MD).

Study	Correlation coefficient between RNFL and MD
Sihota R et al. 2006 [16]	0.626 (p<0.001)
Ajtony C et al. 2007 [17]	0.718 (P=0.01)
Horn FK et al. 2009 [18]	0.75 (p<0.001)
Cvenkel et al. 2011 [19]	0.549 (p<0.001)
Kaushik S et al. 2011 [20]	0.560 (P=0.005)

**The present study is in agreement with the study of Bobrow, who also found a negative correlation (-0.68) between DDLS or Vertical CDR and MD [21]*

associated with a 19% increased risk of visual field progression[23]. This can explain why the present study observed vague correlation between OCT and Visual Field changes. Nevertheless, progression of visual field disorder in glaucoma is usually slow. The result of this study demonstrates that functional loss in glaucoma relates well with OCT measurement of peripapillary RNFL. Our knowledge concerning the functional/ structural relationship in glaucoma is progressively increasing. So, we can also diagnose a case of POAG by OCT before visual field changes in perimetry (Pre- perimetric glaucoma) because RNFL defects proceed the development of datable optic disc and visual field changes[24,25,26].About 40% of retinal ganglion cells will be damaged by the time the VF changes are first manifested[27,28,29.30].

CONCLUSION:

Ophthalmologists still have to fight a grim battle with the problem of glaucoma. Most frequently involved topographically corresponding sector of VF

(Supero-nasal and Inferonasal) and peripapillary RNFL (Inferotemporal and Supero-temporal) may represent common investigation aimed at exploring the diagnostic ability of the combination of functional and structural test, which has already been proved to be highly promising for more accurate detection of early glaucoma. However, long term studies are needed to be conducted involving considerations for visual field parameter changes in Perimetry and optic nerve changes in Optical Coherence Tomography.

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Learnings for Best Practices of Critical Value Alert in Laboratory Quality Management System

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Original Research Article

ABSTRACT

A cross-sectional study was conducted at a tertiary health care centre in India during year 2018 to 2019 to provide a standard for good laboratory practice, to increase the clinical effectiveness, patient safety and operational efficiency and designing better and more evidence-based systems for timely notification of laboratory results. The entire data was obtained from reports generated by hematology and clinical pathology laboratory recorded in critical call back log register. These laboratories reported 86727 critical values out of 394213 performed test. The majority of critical callbacks (83.8%) resulted from testing performed in the Hematology. The most common called back were Hemoglobin and Total WBCs count. We recorded maximum 52% call back from inpatient department followed by emergency department 35% and outpatient department 13%. The mean time between entering value in the critical callback register and conveying the information to the patient location or ordering clinician was 60 minutes for IPD, 120 minutes for OPD and 30 minutes for ED. The study inferred that each laboratory must have at a modus operandi to alert critical results.

KEY WORDS: critical alert value, hematology, patient safety

INTRODUCTION:

The concept of critical value was introduced more than 46 years ago by Lundberg and has been widely adopted as a standard of good laboratory practice. It suggests that a result which indicates that patient is in imminent danger unless therapy is initiated immediately[1,2,3]. CAP (College of American pathology) has made critical value reporting as a part of requirement for accreditation. Health and safety in clinical laboratories is becoming increasingly important subject currently. The preparation and approval of critical alert value list should be done in consultation with the concerned hospital board/clinician's panel and it is essential to discuss the possible mode of implementation of the same with local facilities[4].

Critical value reporting parameters may be considered an important laboratory outcome measurement because they reflect clinical effectiveness, patient safety and operational efficiency. For the critical value reporting process to be effective, the organization must understand and address the variables involved in critical results reporting with opportunities for process improvement in the process. This information is not readily available in the literature. Most reports have analyzed only a few analytes for short periods or have reviewed a small number of critical values in a number of different institutions [5,6,7].

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MATERIALS AND METHODS:

This cross-sectional study was conducted in Central Pathology Laboratory (Hematology and Clinical pathology section) of NKP Salve Institute of Medical Sciences and Research Centre and Lata Mangeshkar Hospital, Nagpur, Maharashtra, India during January 2018 to December 2019. All major medical and surgical specialties are supported by its 1000 bedded hospital inclusive of pediatric care, obstetric care, extensive primary care and specialist outpatient practices. During the study period the laboratories performed 3,94,213 reportable tests, of which 53% were for inpatients, 13% for outpatients and 35% for emergency department (ED) patients.

The critical callback list for hematology and clinical pathology laboratory used in institutional set

Table 1: Critical values in haematology & clinical pathology.

Sr.No	Parameters	Critical Values
1.	Hemoglobin	< 5gm/dl
2.	Total WBC Count	<20,000 & <4000/cmm
3.	Platelet Count	<50,000/ul
4.	Presence of Blasts Cells.	Present
5.	Presence of Malaria Parasite	Present
6.	Positive Malarial Antigen	Positive
7.	Urine Ketone bodies	Positive
8.	Absolute Neutrophils Count	<1800 /cmm
9.	Immature : Total Neutrophils Ratio	>0.2(Pristine PT)
10.	CSF – Cell Count	>5Cells
11.	INR	>5.0

Table 2: Evaluation of critical values by clinical case areas.

Sr.No	Clinical Areas	No of Critical Call Back
1.	IPD (Inpatient Department)	45098 (52%)
2.	OPD (Out Patient Department)	11275 (13%)
3.	ED (Emergency Department)	30354 (35%)
4.	TOTAL CRITICAL ALERT	86727 (22%)
5.	Gross total number of test result	3,94,213

Table 3: Evaluation of critical callback in haematology & clinical path laboratiries.

Laboratory	Haemat Section	Clinical Path Section	Total
Critical Call back	72677 (83.8%)	14050 (16.2%)	86727

up with deputation of informer personnel (i.e. Resident Doctors, Lab Technicians) usually ensures validation of result by repetition of test or by recalibration of parameter, if necessary and/ or by checking quality control result (Table 1). If the result is in the critical value (upper or lower), the lab personnel would communicate with the responsible caregiver [Consultant, Registrar, Medical Officer, Nurse and Operations associate (i.e. Clerical staff who perform clinical support function)] to inform the values on phone. At the same time he/she will also inform the result to the concerned laboratory doctor (i.e. consultant, senior doctor and in charge). The outpatient critical value is informed to responsible consultant or his/her medical assistant (i.e. medical officer). The laboratory personnel records details of critical call back [parameter, critical value, informed & informer person, date, time (i.e. Result generation, information & Convey interval)] register.

All data were retrieved from reports generated by hematology and clinical pathology laboratory that has been recorded into critical call back register. The observed parameters were evaluated by using descriptive statistical analysis by Microsoft office Excel 2007 software.

RESULTS:

During the study, hematology and clinical pathology laboratories reported 86727 critical values and 3, 9 4,2 1 3 test results reported during the period of two years. Therefore, tests with critical values represented approximately 22% of the total test results. (Table 2) The majority of critical callbacks (83.8%) resulted from testing performed in the hematology laboratory (Table 3). The clinical pathology laboratory accounted for 16.2% of critical callbacks. The analytics most commonly called back for hemoglobin were (31222 results; 36% of critical results) subsequently TLC 23850 (27.5%) of critical call back, Urine Ketone (13009 results; 15% of critical results) and platelets 11708 (13.5%) of critical callback (Table 4).

We have recorded maximum 45964 (52%) call back from inpatient department (IPD) followed by

Table 4: Evaluation of critical values by parameters.

Sr.No	Parameters	Number & Percentage of Critical Call Back	
1.	Hemoglobin	31222	(36%)
2.	Total WBC Count	23850	(27.5%)
3.	Platelet Count	11708	(13.5%)
4.	Prescience of Blasts Cells	434	(0.5%)
5.	Prescience of Malaria Parasite	1041	(1.2%)
6.	Positive Malarial Antigen	780	(0.9%)
7.	Urine Ketone bodies	13009	(15%)
8.	Absolute Neutrophils Count	2602	(3%)
9.	Immature : Total Neutrophils Ratio	520	(0.6%)
10.	CSF – Cell Count	1041	(1.2%)
11.	INR	520	(0.6%)
	Total	86727	

Table 5: Frequency of call back in each shift.

Shifts	Early Morning	Afternoon	Evening	Night	Total
Critical Call Back	39894 (46%)	38854 (44.8%)	6938 (8%)	1041 (1.2%)	86727

Table 6: Evaluation of critical values by tat (turn around time).

Care Areas	TAT (Turn Around Time)		
	Minimum	Maximum	Mean
IPD	30	90	60
OPD	20	180	120
ED	15	45	30

emergency department (ED) 30,354 (35%) and outpatient department (OPD) 11275 (13%) (Table.2). Critical value calls were maximum 39894 (46%) in morning shift and minimum 1041(1.2%) in night shift (Table 5).

The turnaround time for each critical value assessed for correctness and appropriateness of critical value reporting. Turnaround time is the time period between receiving of a sample and generation of report. The mean time between entering value in the critical callback register and conveying the information to the patient location or ordering clinician were 60 minutes (30-90 minutes) for IPD, 120minutes (60-180 minutes) for OPD and 30 minutes (15-45 minutes) for ED as analyzed for 86727 critical values (Table 6).

Gap in critical value reporting and report generation time reveals certain fallacies inclusive of following: (a) There is deficiency of modalities and directives for updating of test performed and information to outpatients (OPD) (b) Testing ordered on requisitions in the form lacking the name of the test ordering clinician and ordering location.(c) It is also found that tests performed in settings (viz. emergency test places) where technicians are continuously present. (viz. coagulation study) hence the critical alerts called back were faster than tests performed in other areas (.i.e. Routine test zone). The information was useful to put into practice all measures to improve critical value reporting in all areas of the laboratory in the form of LIS, HMIS etc.

DISCUSSION:

Study period with existing assets, provides a comprehensive view of the critical value reporting procedure at tertiary care center in addition to the details about scope, volume, timing and operational aspects of critical value reporting. Many parameters are applicable to a variety of settings. This analysis provides a framework for comparison and process improvement required.

Increasing workload: Clinical laboratory makes it important to achieve efficient use of laboratory resources to maximize clinical benefits. Spreading out of critical callback lists to include testing that does not organize the significant factor of the 'impending risk'

standard may reduce the necessity of a critical value call and direct to needless interruptions for clinicians viz. critical value calls for high INR levels will not be of clinical value for patients receiving heparin in cardiac operations. In addition, there are many clinical settings (chemotherapy, malignancy) in which the 'critical' test result is expected and reporting of this value may not contribute to improved patient care. Hence, there is felt need of modified upgraded LIS, HMIS and Server with traceable Cloud based System having confidentiality.

Communication Devices: Communication by technicians is a costly practice in terms of the resources required to complete the phone calls and document the process, in favor to this reason, it is helpful to try and reduce the number of phone calls by careful review of the critical values list. In addition to determining which tests are to be included in the critical values list, another important strategy is to examine the consequences of changing the boundaries for critical value reporting. These boundaries must be defined in consultation with clinicians. Small changes in critical value reporting parameters may result in the addition or loss of thousands of phone calls for the laboratory staffs. Its better to have link with LIS and HIMS to display the color code of abnormal biological reference values and Critical Values must be précised by joint venture of Lab & Consultant authorities to avoid gap between result generation time and information time, which will become in Critical alert as well as TAT maintenance under guideline of accreditation.

***Critical values of OPD*:** This is an exceptional task to report clinicians. The strongest correlates of delayed reporting of critical values were the samples of an outpatient (i.e. OPD). Outpatient critical values are required to communicate to the responsible clinician who is not traceable because both are having different approaches in various practices for decision of patient coverage. They are not like inpatients. There is no fixed patient location that can be called on mobile number for tracking through SMS/What sup. So OPD patients required location communication of both patients and consultant to inform /to display with color code on communication devices by upgrading APP provision of Web site based cloud system with confidentiality.

Non accessible Authority/Scrawled/Missing: Another factor observed during this study causing delay for outpatients was scrawled or missing patients information. It is also found that recent improvements in the critical value communication times have coincided with increased awareness of critical value monitor working with outpatient practices to improve communication between the laboratories and the outpatient care centers by using HIMS, App, and Website based cloud system with confidentiality. It must be upgraded with accessibility and traceability via communicating devices. Next contributor to delay in outpatient critical value reporting is the heterogeneity of the outpatient population having specimen receipts from health centers (i.e. RHTC), clinics (OPD), urgent care centers (satellite center), dialysis centers, casualty and physicians offices. Each of above areas are liable to have a different call schedule, answering service and cross-coverage procedure, making reliable communication with the responsible licensed caregiver is still not easy as they may shift to emergency services provider zone i.e. IPD, MICU, NICU, PICU, ICCRU, ICCU for emergency services. The nature of outpatient specimen transport and processing often results in outpatient test results being generated in the evening when the outpatient clinic or physician's office is closed. The laboratory must have a mechanism to determine on-call coverage and work with outpatient practices to improve the communication processes.

Up-gradation (potential solution) of critical value reporting technique: The use of information technology (i.e. HIMS) to automatically communicate with the responsible provider has proven to help reduce critical value reporting time in controlled settings.[8,9] For implementation of automated critical value reporting, interfaces from the LIS to technologies that facilitate bidirectional communication (such as e-mail, whatsapp or 2-way pagers) need to be developed. An important component in such a system is the ability of the automatic reporting system to reliably determine the identity of the responsible provider. At larger medical centers, this task can be challenging because there may be different coverage lists, tests ordered by consultants unknown to the primary caregiver and patient transfers to different locations. An electronic reporting system potentially could create dangerous delays in communication if not properly implemented. The system needs to have a 'recognition' function such that the laboratory can ensure that the responsible caregiver has received the result[10]. Electronic systems also requires an intensification procedure so that lack of acknowledgment of the critical result prompts an alternative approach for communication in the form of APP provision of Web site based cloud system with confidentiality to assess openly any service provider

and beneficiaries directly/ vice versa [8,910].

Up gradation of LIS: Rules-based logic can be applied to laboratory values to build alerts that take into account not only the result value, but also other related results, a change in the current test result from previous results (e.g. delta checks, Sigma check), patient demographics, ordering provider rejection follower and other parameters to customize for alerting patient's condition and the needs of clinical team for notification viz. many oncologist do not want to be notified regarding patients with neutropenia. The ability to provide a physician specific critical values list could eliminate a large number of unnecessary critical value calls. These systems, when interfaced with automated alerting systems, will have the potential to improve patient safety and provide further frameworks for susceptible critical value reporting.

CONCLUSION:

Laboratory must have clearly defined and technically enriched procedure for informing and alerting all concerned systematically for critical results. An agreement should be made with clinicians to establish a specific list of critical limits according to the type of patients and the relevance of laboratory test. Laboratories have a system of critical call back system in laboratory with accreditation, which must be followed for ensuring patient's safety. The entire scenario can be upgraded through website based cloud system with password protected access of results by authorities and beneficiaries. This will reduce laboratory errors and progressively improve quality, efficiency and outcomes. However, extensive follow-up, monitoring with continued documentation are necessary to ensure long term success of quality management systems.

It is hence inferred that the critical value reporting is crucial for patient safety as standardization of such practice would be beneficial. It is also essential for effective use of the existing resources and it also creates professional responsibility. Regular quality assessment review meeting with technical staff and strict adherence to the set norms are thus the key to achieving the goal of quality management in laboratories.

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Assessment of Important Biochemical Parameters in Urban Hypertensive Adolescents

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Original Research Article

ABSTRACT

The prevalence of Hypertension among youth is increasing and hence its early diagnosis in adolescent population, detection of metabolic defects, and their clinical management are important. Hence, various biochemical parameters including Fasting glucose, LFT, RFT, Electrolytes, Lipid profile, Hs-CRP and Homocysteine) among HT- Adolescents (HT-A) and Control Adolescents (C-A) were assessed. The study revealed decrease (10-20%) in the biochemical parameters viz. ALP, BUN, UA, Calcium, and the Electrolytes of confirmed HT–adolescent subjects of age group 13-18 years, while Hs-CRP (41.3%) and Homocysteine (75.6%) levels were found elevated.

KEY WORDS: adolescents, homocysteine, hs-CRP, hypertension

INTRODUCTION:

Hypertension is fourth most prevalent killer disease in the world[1,2]. This is silent killer- disorder by itself and can lead to stroke, renal damage, congestive heart failure and similar disorders. Hypertensives mostly suffer from these complications and organ damage, which initially remains undetected and untreated[3,4].The prevalence of Hypertension in the young Indian population, age group <40 years, is also increasing.

Hence it is important to diagnose these conditions in the early age and to try to reverse the high blood pressure in order to avert damage of the target organs[4,5]. The study was focused on the adolescent group.

MATERIALS AND METHODS:

The study was conducted on confirmed young essential Hypertensives of 13-18 year, not on any antihypertensive medications, attending Hypertension OPD of speciality clinic of General Medicine Department of K.E.M. Hospital, Parel, Mumbai, the Tertiary Care Hospital in Mumbai. The population of students of local School from 8th standard to 10thstandard and Junior College children studying up-to age 18 years were considered for screening (N= 5000). The ethical permission was obtained by the local Institutional Reference Committee (IRC-I) for the study and all other permissions were taken. Their relevant anthropometric data were collected.

The comparative aspects of important biochemical parameters (Fasting glucose, LFT, RFT, Electrolytes, Lipid profile (all on Autoanalyzer), hs-CRP (Nephalometric Method) and Homocysteine (Nephalometric Method) from fasting blood samples of HT- Adolescents as Test group and Normal Adolescents as Control group were studied. The results were statistically analysed (N=10) using Students 't' table.

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RESULTS:

The observations of this study are useful in understanding the trends of studied parameters of Hypertension in the adolescent-groups. The BMI was higher in the Test group (Table 1). This might be due to higher obesity, less physical activity in this group. All members in this group were Male.

The study conducted on confirmed male Hypertensive-Adolescents noted marginal decrease (10-20%) in biochemical parameters viz. Fasting glucose, Total proteins and Albumin contents, the enzymes (Alkaline phosphatase (ALP), Aspartate aminotransferase (AST) and Alanine amino

Table 1: Anthropometric parameters in Test and Control Adolescents.

Parameters	Test Group	Control Group	p-value
Weight (Kgs)	52.73+7.80	53.10 + 8.10	N S
Height (Cms)	147.7+ 25.0	150.0+23.	N S
BMI	34.4+2.1	32.4+ 2.5	N S
Blood Pressure			
Systolic (mm of Hg Column)	134+ 16	114 + 4.0	<0.001
Diastolic (mm of Hg Column)	94+ 6	80 +6.0	≤0.001
Pulse	86+10	72+4	≤0.001
Fasting Plasma Sugar (mg%)	105 .5+ 10.5	114 +20	N S
Total Protein Contents (g%)	6.9+ 1.2	7.2+ 0.5	N S
Albumin Contents (g%)	4.6+0.8	5.0 + 0.8	N S
BUN (mg/%)	28 .83+ 4.0	25.0 + 4.7	N S
Creatinine (mg/%)	0.7+ 0.2	0.6+ 0.2	N S
Uric Acid (mg/%)	3.38+1.7	3.20 + 0.8	N S
Calcium (mg/%)	10 .5+0.8	9.9 +0.5	N S
Phosphorous (mg/%)	3.9 + 0.5	3. 6 + 0.7	N S
Alanine Aminotransferase ALT (IU/L)	39.33+ 7.5	35.33 + 6.9	N S
Aspartate Aminotransferase AST (IU/L)	31.43+ 7.0	36.10 +4.0	N S
Alkaline Phosphatase ALP (IU/L)	105+18.0	117.0 + 20.0	N S
Sodium (mEq/l)	133 .66	140 .50	N S
Potassium(mEq/l)	4.1+0.3	4.5 + 0.2	N S
Chloride(mEq/l)	97.6 +1.2	100 .5 +3.2	N S
Total Cholesterol (mg/%)	180.0 + 18.0	168.0+ 13.0	N S
Triglycerides (mg/%))	135.4+10.3	130.4 + 12.2	N S
HDL-Cholesterol(mg/%))	37.6 +6.2	45.5 +10.2	N S
LDL-Cholestrerol (mg/%)	129.5+16.7	100.5+ 11.2	N S
VLDL-Cholesterol (mg/%)	33.0 + 11.6	23.0 + 12.8	N S
Total Bilirubin(mg/%)	1.4 +0.3	1.4 +0.2	N S
Direct Bilirubin(mg/%)	0.6+0.1	0.5 +0.2	N S
Homocysteine(mg/%)	23 .8 +3.4	13 .2 +2.7	<0.001
Hs-c-Reactive Protein (Hs-CRP)(mg/l%)	14 .13+2.1	10 .1 +2.3	<0.001

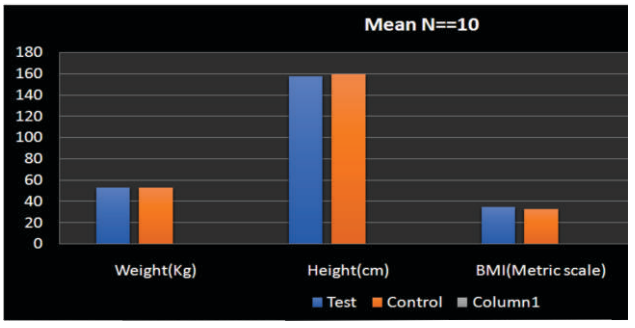
transferase (ALT), Blood Urea Nitrogen (BUN), Uric Acid (UA), Creatinine, Calcium, and Electrolytes (Sodium, Potassium and Chloride contents) of confirmed Hypertensive adolescent subjects. These values were within normal range and were statistically insignificant.

In case of lipid profile, comparative analysis of the Total Cholesterol contents and its other fractions (HDL-C, LDL-C) along with TGs exhibited insignificant decrease in the test group. In fact, the higher values are noticed in respective control groups.

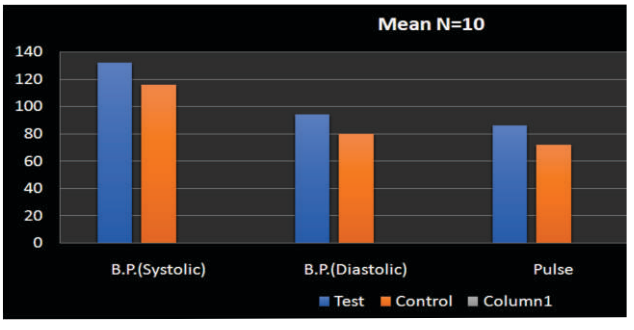
The elevations were observed in hs-CRP (41.3%) and Homocysteine (75.6%) levels in Test groups. It revealed early alterations in the Hypertensive adolescents. These were statistically significant in either case. It indicated that Homocysteine and Hs-CRP are the parameters which could be considered as the bio-markers of Hypertension in young hypertensives. Early diagnosis and detection of metabolic defects of Hypertensive patients are crucial. The causes and the metabolic changes in the disorders may be obscure, but later their effects are dangerous and fatal (4,5).

The study included confirmed Hypertension patients between 13 years to 18 years. Homocysteine and hs-CRP are the parameters which can be considered as the biomarkers of Hypertension in young hypertensives, while other routine parameters failed to show any significant alterations. The observations were useful in checking the trends in the adolescent-groups. The BMI was higher in the Test

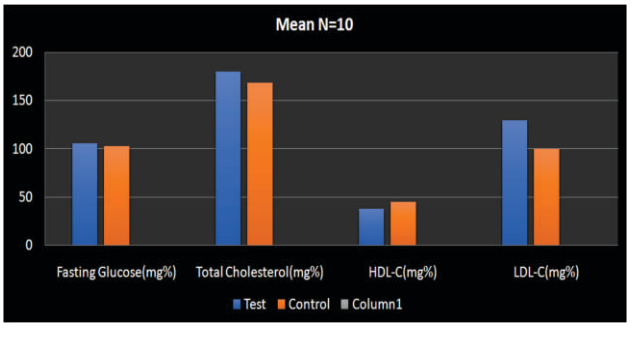
Graph 1: Anthropo-physical parameters in HT-Adolescents.



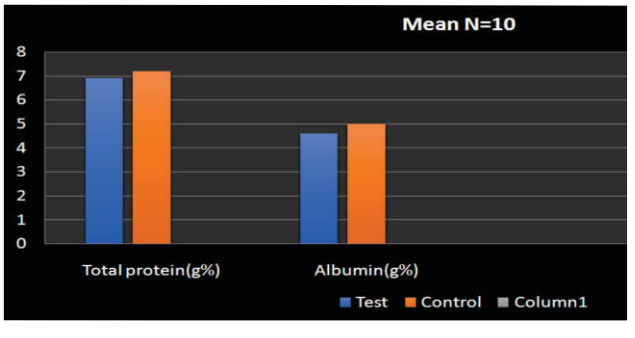
Graph 2: B.P. & Pulse of the HT-Adolescents.



Graph 3: Important Biochemical Parameters.

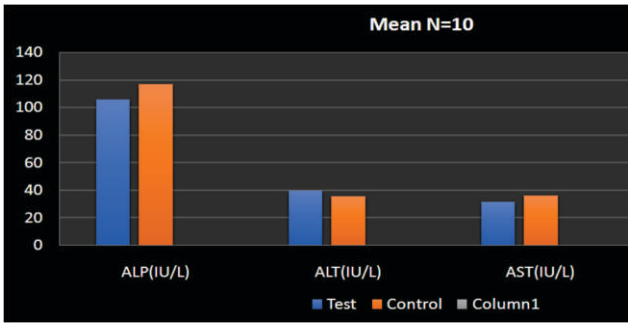


Graph 4: Serum Total Protein & Levels of Albumin..

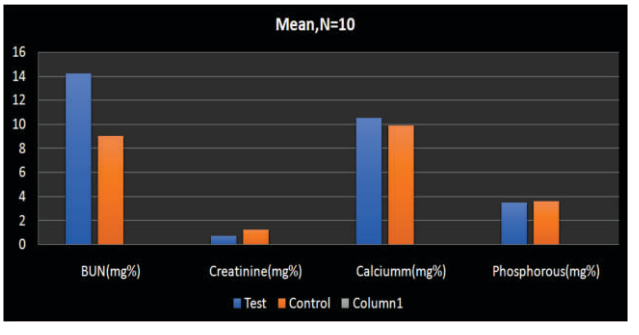


group (Table 1). This might be due to higher obesity, less physical activity in this group, especially in males. It could be associated with stess in the academics like assessment patterns and competitiveness at various levels. Various all India-entrance examinations, their results and selection of career, threat of failure, and

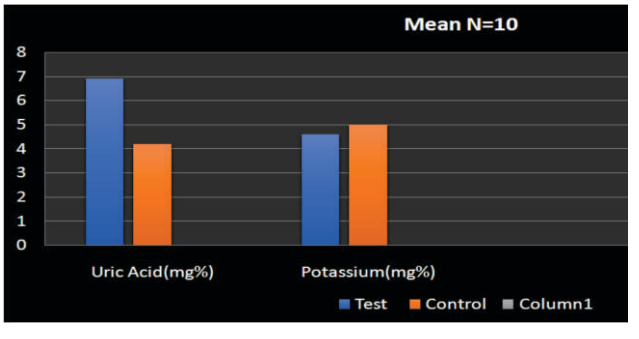
Graph 5: Important Enzymes in HT-Adolescents.



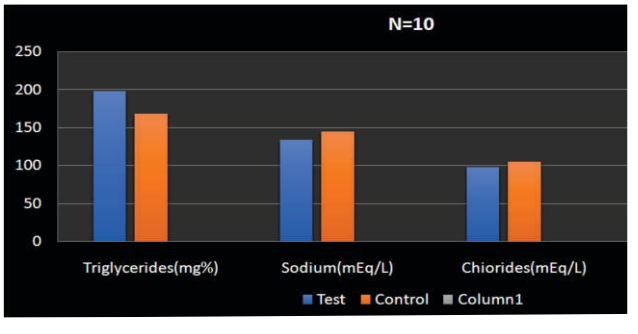
Graph 6: Levels of BUN, Serum Creatinine, Calcium, Phosphorous.



Graph 7: Levels of Serum uric acid and Potassium.



Graph 8: Levels of TGs, Sodium and Chlorides.

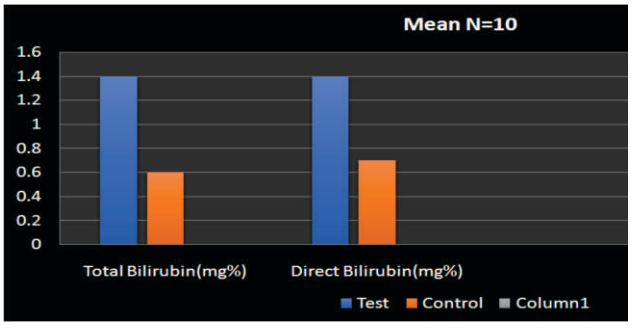


coping with learning of new professional courses may be influential factors.

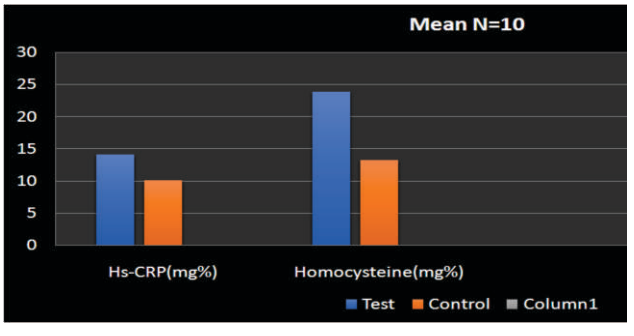
DISCUSSION:

Around 40% adult population of more than 40

Graph 9: Total Bilirubin and Direct Bilirubin in Test & Control groups.



Graph 10: Hs-CRP and Homocysteine Levels.



years suffer from Hypertension worldwide. Younger population is also vulnerable to Hypertension. Out of 5000 screened adolescents, 10 male adolescents were clinically confirmed as the essential hypertensives (1,2,5-7). The prevalence of 1:500 and only of male population are the facts needed to be confirmed with large data[3,4].

The fasting blood samples of the group were analysed for the important biochemical parameters(Graphs 1,2). The changes in height, weight and BMI were statistically insignificant in the Hypertensive group which might be due to variable and fast growth in this phase of life[8,9]. There was 10-20% decrease in the contents of Blood glucose, Total Proteins & Albumin of the Test group (Graphs 3, 4), which were statistically insignificant. Total Cholesterol and its fractions along with TG levels also exhibited insignificant effect in the essential Hypertension group (Graph 3, 8). The hypertension in the Test group could not be correlated to these parameters in early stages. Other researcher could establish such correlation in the adult hypertensives [7,8,9].

In the current study, the liver enzymes, minerals and electrolytes levels were within normal range in early Hypertensives (Graphs 5, 6,7). This indicated metabolic scenario among hypertensives in the beginning. Later on, with involvement of the other organs, these parameters showed elevated levels which was also discussed by other research scholars [9,10,11 & 12]. Significant changes were observed for Homocysteine and Hs-CRP levels (Graph 10). These parameters also formed early markers of adolescent hypertension. This has been confirmed in the studies of the other adult hypertensive cases[3,13]. The increased homocysteine contents could be the result of deficiency of Vitamin B12 in this population. In this case it would be worth assessing Vitamin B12 contents and trying supplementation of same in the urban essential hypertensive adolescents [13,14,15].

CONCLUSION:

This study identified hypertension in urban adolescents although the prevalence is low. There is need to keep regular check -ups for this population as they are subjected to number of stresses in this age-group. The Homocysteine and Hs-CRP profiles from large data would be of great help to confirm and assess the causes of hypertension for this population.

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Serum Electrolyte Levels among Hospitalized Cases of Acute Coronary Syndrome in A Tertiary Care Hospital

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Original Research Article

ABSTRACT

Hypomagnesemia is common in hospitalized patients, especially in the elderly with acute coronary syndrome and/or those with chronic heart failure. The present study was carried out in the department of Medicine, Peoples Medical College and research centre, Bhopal from November 2017 to October 2018. Sixty patients of acute coronary syndrome diagnosed by clinical examination and ECG criteria were included in the study. Arrhythmias and conduction blocks were noted on continues ECG monitoring in ICU. These patients were analyzed for serum magnesium, calcium, sodium and potassium concentration on admission. It was found that serum magnesium and serum sodium are significantly changed between cases and controls($p < 0.05$) but serum calcium and potassium are not significantly changed ($p > 0.05$).

KEY WORDS: acute coronary syndrome (ACS), non ST elevation myocardial infarction (NSTEMI), ST elevation myocardial infarction (STEMI), unstable angina (UA)

INTRODUCTION:

The word acute coronary syndrome (ACS) means any group of clinical symptoms compatible with acute myocardial ischemia (AMI) and covers variety of clinical conditions including unstable angina (UA), non ST-segment elevation myocardial infarction (NSTEMI) and ST-segment elevation myocardial infarction (STEMI). Unstable angina and NSTEMI are intricately related conditions having similar clinical presentations are same although differing in severity only[1,2,3]. A diagnosis of NSTEMI is made when the ischemia is severe enough to cause myocardial damage that results in the release of a biomarker of myocardial necrosis into the circulation like troponins T or I, or muscle and creatine kinase [CK-MB]. Whereas the patient is assumed to have experienced UA, if no such biomarker is detected in the blood hours after initial complaints of ischemic chest pain. Unstable angina shows 3 principal presentations: (1) resting angina (usually lasting >20 minutes), (2) new-onset (<2 months previously) severe angina, and (3) a pain character which is increasing in intensity, duration, frequency, or any combination of these factors[4,5,6].

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MATERIALS AND METHODS:

The study was conducted in ICU ward in department of Medicine, Peoples hospital, Bhanpur Bhopal. Ethical approval for the present study was obtained from the institutional review board. This study included clinically diagnosed 60 case of acute coronary syndrome and 60 age and gender matched healthy controls selected from general population for estimating serum magnesium, serum calcium, serum sodium and serum potassium concentrations. Matching was done after completion of study. Electrocar-diographic recordings of all these patients were taken.

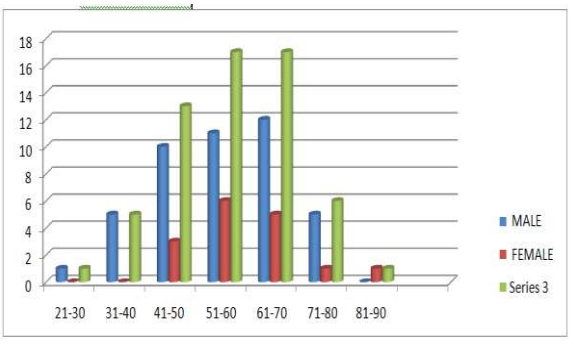
RESULTS:

Age of patients in study group ranged from 21 - 90 years of 60 patients. Forty four patients (73.33 %) were Male, whereas sixteen (26.66%) were Female. Male Female ratio was 3:1. The incidence of ACS was more in males as compared to females. Both in males & females, increased incidence of ACS was found between 51 - 70 years (50.96%) (Table 1, Graph 1) The age of controls ranged from 21-90 years. Forty four patients (73%) were males & 16 patients (27%) were females with male: female ratio of 3:1. 46 patients (77%) out of 60 cases belonged to age group of 41-70 years. (Table 2, Graph 2).

Out of 60 patients STEMI had developed in 21 patients (35%). 20 patients (33%) developed

Table 1: Age and Gender distribution of Study Group.

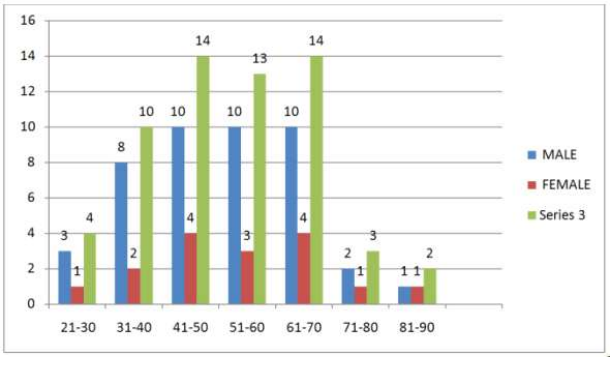
Age Group	Male	Female	Total
21 - 30	1	0	1
31 - 40	05	0	5
41 - 50	10	3	13
51 - 60	11	06	17
61 - 70	12	05	17
71 - 80	05	1	06
81 - 90	00	1	01
Total	44	16	60



Graph 1: Age and Gender distribution of Study Group.

Table 2: Age and Gender distribution of Control Group.

Age Group	Male	Female	Total
21 - 30	03	01	04
31 - 40	08	02	10
41 - 50	10	04	14
51 - 60	10	03	13
61 - 70	10	04	14114
71 - 80	02	01	03
81 - 90	01	01	02
Total	44	16	60



Graph 2: Age and Gender distribution in Control Group

unstable angina. 19 patients (32%) developed NSTEMI. (Table 3, Graph 3) Out of 60 patients of ACS, 29 patients (48.33%) had hypocalcemia, 19 patients (31.66%) developed Normocalcemia, 12 patients (20%) developed hypercalcemia. Out of 29 patients of hypocalcemia, 10 patients are of STEMI (16.67%), 19 (31.66%) patients are of unstable Angina and NSTEMI. Out of 12 patients of hypercalcemia, 6 patients are of STEMI (50%), 6 patients are of unstable Angina and NSTEMI (50%).(Table 4, Graph 4)

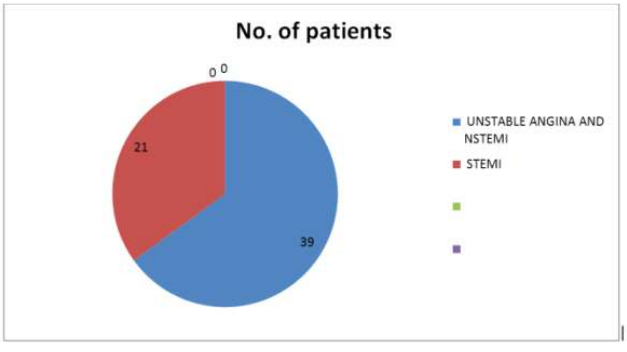
Out of 60 patients of ACS, 56 patients (93.33%) had normal potassium level. Only 3 (5%) patients had Hypokalemia. Out of 21 patients of STEMI, 2 patients (9.52%) had hypokalemia and 18 patients (85.71%) had normokalemia. Only one patient developed hyperkalemia. Out of 39 patients of unstable angina and NSTEMI, 38 patients (97.43%) had normokalemia and 1 patient (2.56%) had hypokalemia. Out of 60 patients of ACS, 29 patients (48.33%) had hypocalcemia. 19 patients (31.66%) developed normocalcemia, 12 patients (20%) developed hypercalcemia. Out of 29 patients of hypocalcemia, 10 patients were of STEMI (16.67%) and 19 (31.66%) patients had unstable Angina and NSTEMI. Out of 12 patients of hypercalcemia, 6 patients had STEMI (50%) and remaining 6 patients had unstable Angina and NSTEMI (50%). (Table 5, Graph 5) Out of 60 patients of ACS, 36 patients (60%) developed hypomagnesemia and 9 patients (15%) had hypermagnesemia. Out of 36 patients of hypomagnesemia, 15 patients (41.66%) had STEMI, 21 patients had unstable angina and NSTEMI (58.33%). Out of 9 patients of hypermagnesemia, 1 patient (11.11%) had STEMI and 8 patient (20.51%) had unstable angina and NSTEMI. (Table 6, Graph 6)

Out of 60 patients of ACS, 15 patients (25%) developed hyponatremia and 45 patients (75%) had normal sodium level. No patient developed hypernatremia. Out of 21 patients of STEMI, 5 patients (23.81%) had hyponatremia and 16 patients (76.19%) had normonatremia. Out of 39 patients of unstable angina and NSTEMI, 29 patients (74.36%) had normonatremia and 10 patients (25.64%) had hyponatremia. (Table 7, Graph 7)

Out of 21 patients of STEMI, 5 patients (23.80%) developed conduction block, 2 patients (9.52%) developed arrhythmias and rest 14 patients (66.66%) did not develop arrhythmias.

Table 3: Distribution of patients of Acute Coronary Syndrome.

Types of ACS	No. of Patients	Percentage %
Unstable Angina and NSTEMI	39	65
STEMI	21	35
Total	60	100



Graph 3: The Distribution of ACS Cases Serum Sodium, Potassium, Magnesium and Calcium Levels in Controls and Cases.

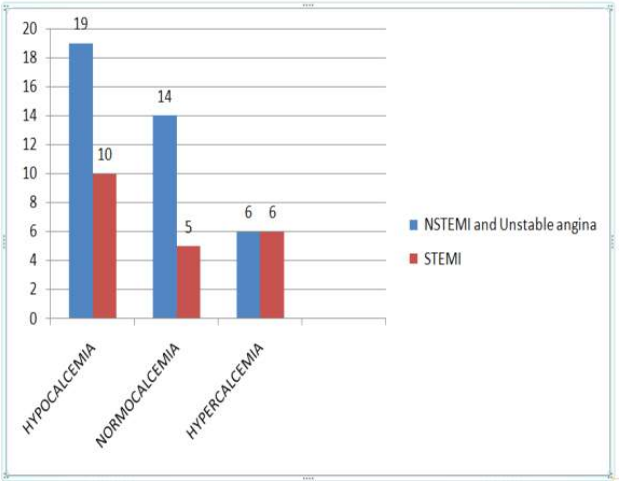
Table 4: Serum Calcium Level in ACS Cases.

S. Calcium Conc. in meq/L	NSTEMI And Unstable Angina	STEMI	Total
Hypocalcemia (< 9 meq/L)	19	10	29
Narmocalcemia level (>9-11 meq/L)	14	5	19
Hypercalcemia (>11 meq/L)	6	6	12
	39	21	60

(Table 8, Graph 8). Out of 21 patients of STEMI, 12 patients (57.14%) had hypomagnesemia and 4 patients (19.05%) had hypermagnesemia. Remaining 5 patients (23.80%) had normomagnesemia. Out of 12 patients (57.14%) of hypomagnesemia, 5 patients (23.80%) had arrhythmia. Serum Magnesium Level in Cases of STEMI with Arrhythmias and Without Arrhythmias were also assessed (Table 9, Graph 9) Out of 12 patients of hypomagnesemia, 3 patients expired (25%). An assessment was done for relationship of hypomagnesemia and mortality (Table 10).

DISCUSSION:

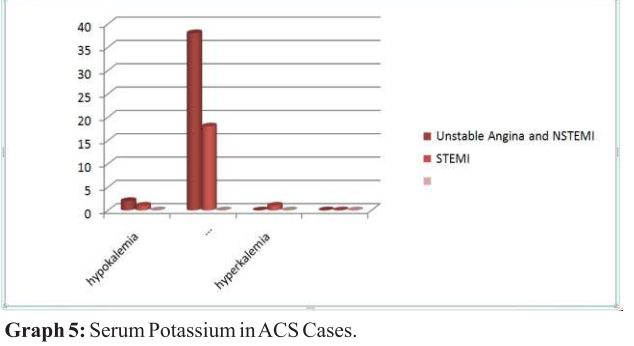
Out of 21 patients of STEMI, 12 patients



Graph 4: Incidence of Serum Calcium in ACS Cases.

Table 5: Serum Potassium Level in ACS Cases.

S. Potassium in Conc. meq/L	Unstable Angina and NSTEMI	STEMI	Total
Hypokalemia (< 3.5meq/L)	1	2	3
Normokalemia (3.5 - 4.5 meq/L)	38	18	56
Hyperkalemia (>4.5 meq/L)	0	1	1
TOTAL	39	21	60

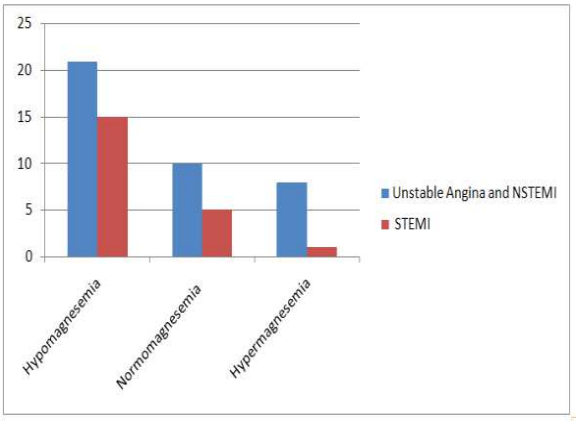


Graph 5: Serum Potassium in ACS Cases.

(57.14%) had hypomagnesemia and 4 patients (19.05%) had hypermagnesemia, remaining 5 patients (23.80%) were normomagnesemia. Out of 12 patients (57.14%) of hypomagnesemia, 5 patients (23.80%) had arrhythmia. Z- test was applied [Z>2 (i.e.,2.346] suggestive of significant relationship between hypomagnesemia and arrhythmias. Out of 21 patients of STEMI 12 patients (57.14%) had hypocalcemia and 3 patients (14.28%) had hypercalcemia, rest 6 patients (28.57%) are having normal calcium

Table 6: Serum Magnesium Level in ACS Cases.

S. Magnesium Conc. in (meq/L)	Unstable Angina and NSTEMI	STEMI	Total
Hypomagnesemia (< 1.7 meq/L)	21	15	36
Normomagnesemia (>1.7 - 2.4meq/L)	10	5	15
Hypermagnesemia (>2.4 meq/L)	8	1	9
TOTAL	39	21	60



Graph 6: Serum Magnesium in ACS Cases.

Table 7: Serum Sodium Level in ACS Cases.

S.Sodium Conc. in meq/L	Unstable Angina and NSTEMI	STEMI	Total
Hyponatremia (< 135 meq/L)	10	5	15
Normonatremia (135 -145 meq/L)	29	16	45
Hypernatremia (>145 meq/L)	-	-	-
TOTAL	39	21	60

level. Out of 12 patients of hypocalcemia, 5 patients (23.80%) had arrhythmia. Statistical Z test was applied and z > 2 [z = 2.67] suggestive of significant correlation between hypocalcemia and arrhythmias.

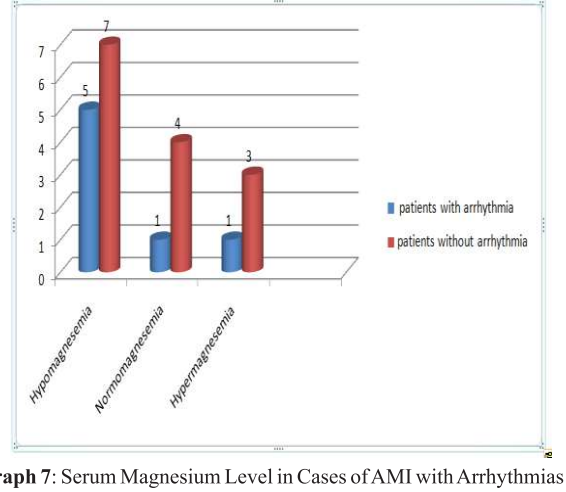
Dyckner T (1978)[7,8] found supraventricular tachycardia in 50% of hypokalemic patients, 40 percent of the normokalemic patients and 45 percent of the hyperkalemia. He also observed atrial fibrillation in 42% of hypokalemic group. Thirty percent of hyperkalemic group had AF. The incidence of atrial

Table 8: Arrhythmias in STEMI.

In AMI Patients	No. of Patients	Percentage
With Arrhythmias	2	9.52
With conduction block	5	23.80
Without Arrhythmia	14	66.66
Total	21	100

Table 9: Serum Magnesium Level in Cases of STEMI with Arrhythmias and Without Arrhythmias.

Serum levels of Magnesium	Patients with Arrhythmias	Patients Without Arrhythmias	Total
Hypomagnesemia (< 1.7 meq/L)	5	7	12
Normomagnesemia (1.7-2.4 meq/L)	1	4	5
Hypermagnesemia (> 2.4 meq/L)	1	3	4
Total	7	14	21



Graph 7: Serum Magnesium Level in Cases of AMI with Arrhythmias and without Arrhythmias.

fibrillation was more in hypokalemic group as compared to normokalemic and hyperkalemic groups.Similarly NodrehaugEJ (1981)[9] noticed that atrial fibrillation was present in 34% of patients. Dyckner T, Nodrehaug EJ and Salmon observed higher incidence of ventricular premature beats and ventricular tachycardia in hypokalemic groups as compared to normokalemic and hyperkalemic groups.

Table 10: Mortality Vs Serum Magnesium Concentration after STEMI.

FATE	Hypomagnesemia	Normomagnesemia	Hypermagnesemia	Total
Death	3	1	1	5
Recovery	9	3	4	16
Total	12	4	5	21

In the present study, maximum patients are in normokalemia group about 19 patients (92.47%). Only 2 patients (9.52%) had hypokalemia and none hypokalemic patients had arrhythmias, suggestive of no significant relationship between hypokalemia and arrhythmias. Dyckner T (1978)[7,8] observed increase number of patients with CHB, 2nd degree heart block in hypokalemic patients 40% and 16% respectively. Left bundle branch block was equal in hypo and hyperkalemia[8] cases. Right bundle branch block and ventricular premature beats were common in hyperkalemic group. In the present study, none of the hypokalemic patient out of 2 patients developed any type of conduction block. The incidence of conduction block was highest, 5 patients (22.22%) developed conduction block out of which left bundle branch block (LBBB) was commonest, Others with conduction block had either right bundle branch block (RBBB) or Right bundle branch block with Left Anterior fascicular block (LAFB). None had Left post fascicular block.

Nodrehaug J (1981)[9]demonstrated increased incidence of mortality in females as compared to males after ACS. In the present study, mortality in patients of STEMI with arrhythmia was 44.82%, while mortality in patients without arrhythmia was 13.56%. Mortality in females (33.33%) was more as compared to males 23.80%. Out of 6 female patients with arrhythmias three expired (50%), while out of seven female patients without arrhythmias two patients (28.57%) expired. Overall mortality was 5 patients (23.80%) in 21 patients of STEMI. Mortality in patients of STEMI with arrhythmia was 44.82%, while mortality in patients without arrhythmia was 14.28%, mortality in female (33.33%) was more as compared to males 23.80%. Out of two female patients with arrhythmias one had expired (50%), while out of three female patients without arrhythmias one patients (33.33%) expired. Out of five male patients with arrhythmias two patients (40%) expired, while out of 11 male patients without arrhythmias one patients [9%] expired. When STEMI was associated with arrhythmias, the incidence of mortality was more in both males & female. There is strong association between patients of arrhythmias and mortality. Out of five expired patients, three patients (60%) had hypomagnesemia, whereas one patient (20%) had hypermagnesemia. Out of 12 patients of hypomagnesemia 3 patients expired (25%). Statistical z test [(z = 6.98) > 2] IS suggestive of significant relation between hypomagnesemia and mortality.

Michael Shechter (2003)[10] observed that magnesium therapy in patient of suspected case of AMI reduced incidence of arrhythmias, congestive heart failure, and conduction disturbances compared with placebo (27% vs 40%, p = 0.04; 18% vs 23%, p = 0.27; 10% vs 15%, p = 0.21, respectively). It was concluded that magnesium sulfate should be considered as an alternative therapy to thrombolysis in patients with AMI.

LIMIT-2 (Second Leicester Intravenous magnesium Intervention Trial)[12,13] in a 2316 patient based study inferred statistically significant decreases in mortality, heart failure and dysrhythmia rates when intravenous magnesium was administered to patients with suspected AMI. Moreover, magnesium was associated with a 16% relative reduction (1.9% absolute risk reduction) in all cause mortality over a 2.8-yr follow-up period (p = 0.03). Researchers concluded that the benefits seen in experimental is chemia-reperfusion models translated into real cardioprotective benefit in the clinical setting. As a result, many physicians incorporated magnesium sulfate into their AMI treatment protocols.

These encouraging results were later challenged, when the Fourth International Study of Infarct Survival (ISIS-4)[10,11], a randomized trial of 59050 patients, reported no 5-week mortality benefit in patients treated with magnesium. Because of its large sample size, this study was compelling, and the use of magnesium for AMI was largely abandoned.

CONCLUSION:

It can be concluded from this present study that serum magnesium and calcium are significantly deranged in patients of ACS but are minor factors in development of arrhythmia solely since serum potassium and sodium are mostly in normal range. However, further research is needed to evaluate and confirm these observations.

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Microbiological Profile of Infectious Keratitis Reported in a Tertiary Care Hospital of Central India

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Original Research Article

ABSTRACT

The study identified bacterial and fungal agents causing infectious keratitis among cases admitted with corneal ulcer and conducted antibiotic susceptibility testing of bacterial isolates. Corneal scrapping from all patients with corneal ulcer received at tertiary health care facility were examined using 10% KOH, Gram's staining methods and culture. Bacterial and fungal isolate identification was done by conventional method. Antibiotic sensitivity was performed for bacterial isolates. Staphylococcus sp. was most common isolate followed by Pseudomonas aeruginosa and Streptococcus sp. Aspergillus niger was most common isolate among fungi.

KEY WORDS: bacterial, fungal, keratitis, tertiary

INTRODUCTION:

Corneal blindness is major public health problem worldwide and infectious keratitis is one of the predominant causes[1]. Developing countries have higher burden of keratitis than developed countries. The etiological agents implicated are viruses, bacteria, fungi and protozoa. In developed world viral infections are leading cause for corneal ulcer, where as in developing countries bacteria, fungi and Acanthamoeba are more common. It is therefore important to know the etiological agent of keratitis[2]. Corneal epithelial defect makes it vulnerable to invasion by organisms[3]. The spectrum of bacterial corneal pathogens depends on the local microbial flora and climatic conditions[4]. Therefore this study was conducted to identify the common organisms causing keratitis in our region.

MATERIALS AND METHODS:

This study was conducted in the Department of Microbiology in a tertiary care hospital associated with NKP Salve Institute of Medical Sciences and Research Centre, Nagpur, Maharashtra, India during June 2015 to June 2018. Corneal scrapping from all patients with corneal ulcer were examined using 10% KOH and Gram staining methods. It was inoculated on blood agar, chocolate agar and Sabouraud dextrose agar. Bacterial isolates were identified based on Gram stain, colony morphology and biochemical tests. Antibiotic sensitivity of bacterial isolates was carried out by Kirby Bauer disc diffusion method. Fungal isolates were identified by the rate of growth, colony morphology on Sabouraud dextrose agar and slide culture[1].

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RESULTS:

The present study was conducted in a tertiary care hospital in Nagpur for a period of 3 years. One hundred and fifty three scrapings from corneal ulcer were sent to the Microbiology department from June 2015 to June 2018. Male patients 103 (67%) were more than female 50 (32.67%). Culture positivity for bacterial and fungal culture was seen in 37 samples i.e. 24% (Table 1).

The mean number of corneal scraping per year from 2015 till 2018 was 52. There were no mixed growth. All the 37 isolates were sole growth from corneal ulcers. Gram stain smear detected organisms in 7 scrapping out of 23 bacterial culture positive. 16 were Gram stain smear negative but culture positive. Fungal hyphae was seen in 7 primary smear / KOH mount out of 14 culture positive fungal isolates.

Among bacterial cultures 18 (78%) were gram positive and 5 (22%) were gram negative isolates (Table 2). The most common bacterial isolate

Table 1: Distribution of Isolates (n=37).

S No	Isolate	Number	Percentage
1	Bacteria	23	62.16%
2	Fungi	14	37.84%

Table 2: Distribution of Bacterial Isolates.

S No	Bacterial Isolates	Number	% of Total Isolates
1	MRSA	04	10.81
2	MSSA	04	10.81
3	CoNS	02	05.41
4	Streptococcus pyogenes	04	10.81
5	Streptococcus pneumonia	03	08.11
6	Pseudomonas aeruginosa	05	13.51
7	Bacillus sp.	01	02.70
	Total	23	62.16

were Staphylococcus species accounting for 10 isolate i.e. 50% of total bacterial culture, followed by Streptococcus sp. at 7 i.e. 30.4%, then Pseudomonas sp. at 5 i.e. 21.7%. Most frequently isolated bacteria was Staphylococcus sp. (10), followed by Psuedomonas aerugenosa (5), Streptococcus pyogenes (4), Streptococcus pneumoniae (3) (Table 2). All bacterial isolates were tested for antimicrobial susceptibility testing, using Kirby Bauer disc diffusion technique. All the pseudomonas isolates are susceptible to Imipenem and, Piperacillin & Tazobactum. They show good susceptibility to ciprofloxacin, Amikacin and Tobramycin (Figure 1). Most frequently isolated fungi was Aspergillus sp. Followed by Fusarium sp, Curvularia sp and Mucor sp. 1 isolate was of Acremonium sp (Table 4).

DISCUSSION:

Infectitious keratitis leads to potentially devastating ocular morbidity as corneal epithelium is breached due to trauma, resulting in ulcer for mation[4,5]. It is leading cause of blindness in developing countries[5]. In the present study, male patients were more in number than female which is similar to study conducted by Chittur et al[5]. However Suwal et al reported female predominance in their study[7]. Abubakar et al also shows that females i.e. house wives are more commonly affected[6].

Infectitious keratitis affects people in their productive age group. Trauma is the major predisposing factor causing corneal ulcers. In our study also injury to eye is seen in 32% of patients. It is consistent finding in other studies as well. [5,6,8] Corneal trauma damages the corneal epithelium which make the underlying tissue susceptible to bacterial adhesion, penetration and replication. Plants, metals, plastic parts, fireworks and pencils cause accidental ocular trauma[9].

Culture positivity for both bacterial and fungal isolates combined was 24%, which was lower than other studies from Nigeria (46.8%), Nepal (44.6%), south India (37.5%), Toronto (57.4%) and Mexico city (34%). [4,5,6,7,8] The low culture positivity may be due to use of antibiotic drops prescribed by general practitioners or self medication by patients. In addition, the viral keratitis doesn't show any growth. Improper collection of sample and delay in transport and processing can also affect results.

Gram stain smear was positive for bacteria only in 7 (30%) cases where as 50% were positive for fungal elements/hyphae. Gram positive bacteria isolated in this study was 78%, which is similar to previously reported in other studies. [9,10,12,13]

Staphylococcus aureus with 8 isolates was the most common isolate, followed by Streptococcus species in 7 isolates, followed by Staphylococcus epididimis in two isolates. This is consistent with findings of Chittur et al[5]. Four isolates were of MRSA. Pseudomonas aeruginosa was the only gram negative bacteria isolated, as seen in Saldana et al[9].

All MRSA isolates were sensitive to vancomycin and tetracycline. Only three were sensitive to ciprofloxacin. All isolates were resistant to gentamycin and chloramphenicol. All Methicillin sensitive staphylococcus aureus (MSSA) were sensitive to ciprofloxacin, clindamycin, vancomycin and tetracycline. All isolate of Streptococcus pyogenes are sensitive to vancomycin and erythromycin. Only two isolates were sensitive to penicillin. Streptococcus pneumonia showed good sensitivity to ciprofloxacin, penicillin, erythromycin and clindamycin.

Pseudomonas aerugenosa has good sensitivity to Imipenem and Pipercillin + tazobactum, similar to Yu et al[10]. It has low sensitivity to ceftazidime, a finding similar to Suwal et al[7]. Al Yousuf et al reports Pseudomonas aeruginosa as the most common causative organism in his study and it is associated with use of contact lens [15-18].

Among the fungal isolates, most common was Aspergillus niger i.e. 5 isolates, followed by 2 isolates each of Aspergillus fumigatus, Curvularia sp,

Table 3: Sensitivity of Gram Positive Isolates.

Bacterial Isolates (No)	Ciprofloxacin	Erythromycin	Clindamycin	Tetracycline	Vancomycin	Linezolide
MSSA (04)	4	2	4	4	4	4
MRSA (04)	2	2	3	4	4	4
MRCoNS (01)	1	1	1	1	1	1
Streptococcus pyogenes (04)	3	2	3	NA	4	4
Streptococcus pneumonia (03)	3	2	2	NA	3	3

#MSSA: Methicillin Sensitive Staphylococcus Aureus; MRSA: Methicillin Resistant Staphylococcus Aureus; MRCoNS: Methicillin Resistant Coagulase Negative Staphylococcus

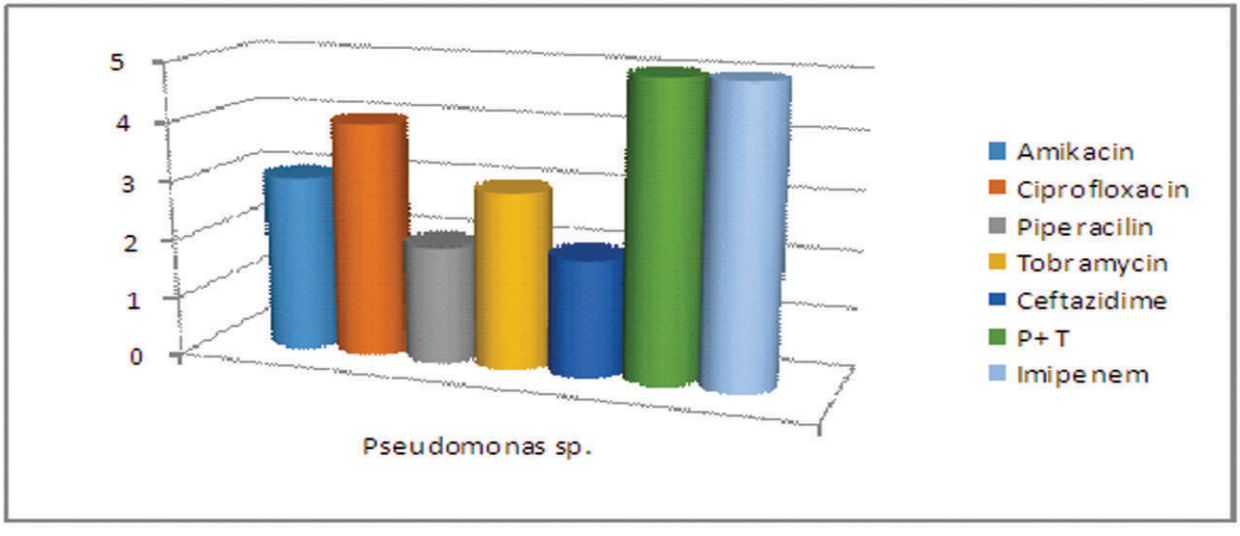

Figure 1: Sensitivity of Pseudomonas sp. # P+T - Piperacillin + Tazobactum.

Table 4: Distribution of Fungal Isolates.

S No	Isolate	Number	% of Total Isolates
1	Aspergillus niger	05	13.50
2	Aspergillus fumigatus	02	05.40
3	Fusarium sp.	02	05.40
4	Curvularia sp.	02	05.40
5	Mucor sp.	02	05.40
6	Acremonium sp.	01	02.70
	Total	14	37.84

Fusarium sp and Mucor sp. One isolate was of Acremonium sp. Our study matches with the results of other studies reporting Aspergillus sp. as most frequent cause of fungal keratitis[2,9,12,13,19].

CONCLUSION:

The study identifies common bacterial and fungal pathogens associated with infectious keratitis and the antibiotic susceptibility of bacterial isolates. Further studies are needed to know the outcome of patients with infectious keratitis. It is also essential to conduct routine surveillance of infectious keratitis for ensuring update on the existing and emerging pathogens.

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Assessment of Bite Force and Masticatory Efficiency in Flexible Partial Dentures: An In-Vivo Study

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Original Research Article

ABSTRACT

The aim of the study is to find the effect of flexible denture base material on bite force and masticatory efficiency in partially edentulous patients. The patients aged between 25 to 50 years were recruited from the Department of Prosthodontics. The inclusion criteria was Kennedys Class III partially edentulous condition, with missing first molar in any one quadrant of mouth, to be restored with flexible RPD with full set of remaining natural teeth. Biting force was measured bilaterally in first molar region, on the dentate side and the prosthetic side using a customized digital gauge. Masticatory efficiency was evaluated bilaterally on the basis of the degree of standardized food sample fragmentation. The mean bite forces in the test side was less (8.04±3.39 N) as compared to the non-test side with bite force with 31.22±3.40 N inferring significant difference between the groups. The masticatory efficiency was also significantly less on the test side as compared to the non-test side. The amount of filtrate material left in the test side was more in comparison to the non-test side. The comparison of maximum bite force and masticatory efficiency have concluded that there is a direct co-relation between their two parameters.

KEY WORDS: dentures, efficiency, masticatory, muscles

INTRODUCTION:

Healthy diet in the form of fresh fruits and vegetables are recommended for humans and such food requires efficient chewing[1]. The word 'efficient chewing' means the breakdown of food with minimum effort and maximum rate of particle size reduction. Chewing efficiency is related to the status of dentition and loss of teeth decreases the chewing efficiency[2].

Assessment of masticatory function in clinical and experimental conditions can be done by measuring bite force and chewing efficiency[3]. Bite force can be defined as the force applied by the masticatory muscles in dental occlusion[4]. In dental research, bite force has been recorded as a variable to assess functional efficiency of various dental procedures like prosthesis, orthodontic treatment or to study effects of deformities and pathologies of the masticatory system and temperomandibular joint[5,6]. A variety of devices with a diversity of designs and working principles have been used to record bite force. It includes portable hydraulic pressure gauge, strain gauge transducers, pressurized rubber tube foil transducers, pressure sensitive sheet and gnthodynamometer[7,8].

According to George A Zarb, average value of occlusal force in natural dentition is approximately 20 kg and 6-8 kg with the denture during chewing and deglutition[9]. There are wide range of variables which influence bite force. They are categorized under individual factors and technique related factors. Individual related factors includes the person's craniofacial morphology, height, age, weight and body mass index (BMI). Technique related factors includes inter-occlusal distance, hardness of biting surface and head position during measurement[10]. It also varies with different means and types of prosthetic restorations and materials.

Injection molded thermoplastic materials like polyamide (PA) and acetal (AC) are alternative to widely used polymethyl methacrylete (PMMA) denture base material. The thermoplastic materials have low modulus of elasticity than PMMA resin and so are flexible in nature[11].

Clinical studies have compared the masticatory performance of patients wearing flexible complete denture (CD) and removable partial denture (RPD) with natural dentition[12,13]. Co-relation between biting force and chewing efficiency in patient wearing flexible RPDs is needed to be evaluated. Therefore the aim of the present study was to evaluate and compare bite force and masticatory efficiency between natural dentition and flexible removable partial dentures. It was hypothesized that flexible denture base material has significant effect on bite force and masticatory efficiency in partially edentulous patients.

MATERIALS AND METHODS:

Participants (n= 15; 9 Male, 6 Female) for the study were recruited from Department of Prostho-

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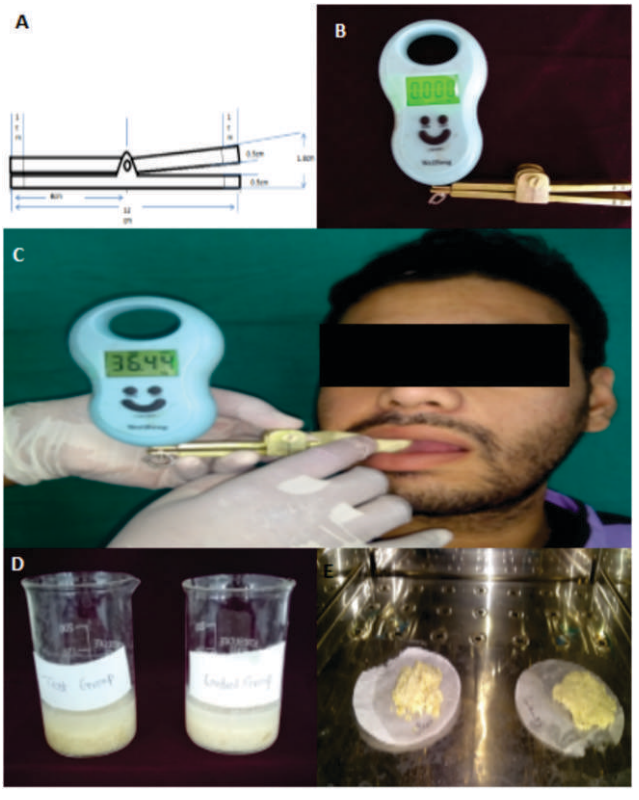


Figure 1: (A) Schematic diagram; (B) Customized digital gauge; (C) Bite force reading displayed on digital meter; (D) Collected chewed food in the biker; (E) Filtrate material dried in hot air oven.

dontics and Crown & Bridge, Hitkarini Dental College and Hospital, Jabalpur, India. The inclusion criteria were patients of age limit between 25 to 60 years with Kennedys Class III partially edentulous condition with missing first molar in any one quadrant of mouth, to be restored with flexible RPD with full set of remaining natural teeth. Remaining natural teeth should show no sign and symptoms, no restorations, carious lesions, no mobility> grade I with vital pulp. The Kennedys Class III partially edentulous condition is used in the study as maxillary posterior teeth is mainly used for chewing and was able to compare with other side of the same patient. Patients should have no systemic condition affecting neuromuscular system, no crossbite or Class I facial profile.

Bite force measurement: Biting force was measured bilaterally in first molar region, on the dentate side and the prosthetic side using a customized digital gauge [Fig. 1(A)]. The customized digital gauge had a digital display unit to show readings [Fig. 1(B)]. It was attached to two stainless steel thongs of dimension 12cm x 1cm x 0.5cm at one end. On the other end of the tongs the subjects were asked to bite with maximum force for 5±1 seconds to record the readings [Fig.1(C)]. The biting force was measured 3 times, with 1 min intervals between the consecutive measurements, and highest value was recorded. All participants were in upright position on dental chair during bite force measurement.

Masticatory efficiency measurement: Masticatory efficiency was evaluated bilaterally on the basis of the degree of standardized food sample fragmentation. Each patient received three portions of 15 ± 1 gram serving of peanuts (Salted peanuts), measured with the aid of a laboratory balance and packed in sealed disposable polyethylene foil bags. The participants were instructed to chew each portion for 20 chewing cycles with a single cycle corresponding to one complete sequence of abduction and adduction of the mandible, along with laterotrusive and mediotrusive movements resulting in crushing and rubbing of the peanuts. The chewed food was collected in a disposable cup. Subjects were asked to rinse twice with water, and the rinsing were added to the cup. Each side was tested three times and the chewed food was pooled for a single measurement for each side of the mouth [Fig. 1 (D)]. The chewed food in the cup was stirred with a glass rod to break up clumps of food and then poured on a stack of sieves 80 mesh U.S. standard sieves. The smaller particles were washed through successive sieves. The particles remaining on sieve were placed on plastic sheets and transferred into 50 C.C. graduated centrifuge tubes. The sieves were rinsed with a cup of water, which were added to their respective tubes. Since no food passed through the 80 mesh sieve, the filtrate was discarded. The tubes were centrifuged for 3 minutes at 1,500 rotation per minute. The volume of the test material (sediment) in each tube was recorded and total volume of recovered food was determined by adding the volumes from all tubes. Filtrate material was filtered with filter paper, remaining material dried in hot air oven for 30 minutes [Fig. 1 (E)] and weighted by laboratory balance and recorded. Greater the quantity of filtrate leftover, less was the masticatory efficiency and vice versa.

RESULTS:

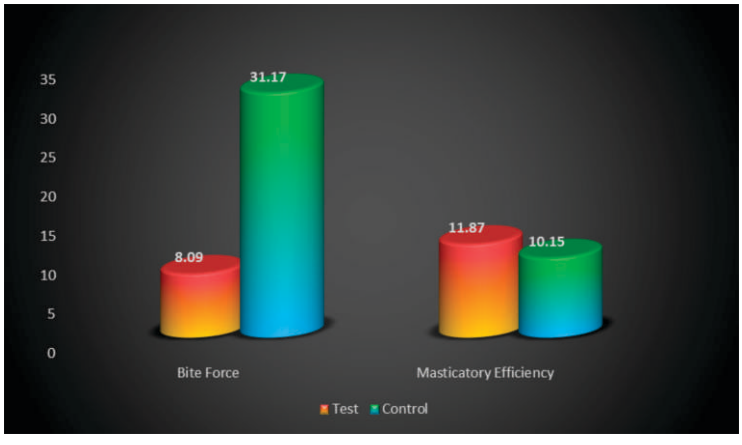
The recorded data was compiled and entered in a spreadsheet computer program (Microsoft Excel 2007) and then exported to data editor page of SPSS version 20.0 (SPSS Inc., Chicago, Illinois, USA). The data shows non normal distribution in Shaprio Wilik test. Hence, non parametric test was applied for

Table 1: Comparison of Bite Force and Masticatory Efficiency between the Flexible RPD (Test Group) and dentate side (Control Group).

Measurement	Group	N	Mean	Std. Deviation	p- value
Bite Force (in Kgs)	Test	15	8.09	3.39	0.001 *
	Control	15	31.17	3.40	
Masticatory Efficiency (filtrate weight)	Test	15	11.87	1.13	0.001 *
	Control	15	10.15	0.69	

*Significant

Graph 1: Comparison of BF and ME between the Flexible RPD (Test Group) and dentate side (Control Group).



quantitative data (mean values). Mann Whitney U test was applied between the mean values of test and control groups for bite force (in kgs) and masticatory efficiency (in grams). Level of significance was set at 0.05.

The bite force (BF) and masticatory efficiency (ME) for test and control group were calculated. Table 1 and Graph 1 shows comparison of BF and ME between the Flexible RPD (Test Group) and dentate side (Control Group). It could be assessed that $p < 0.001$ showed that there was statistically significant difference between the bite force of natural teeth and the flexible partial denture. The mean bite force value and masticatory efficiency was more on dentate side.

DISCUSSION:

Loss of teeth causes extensive change in the oral function, physiology and health. It results in decreased masticatory efficiency, disturbed digestion, malnutrition altered speech and develop esthetic problems. [14] Prosthetic replacement of lost teeth is aimed towards solving the associated problems with lost teeth. According to the clinical condition replacement could be in the form of complete dentures, partial denture, over denture, fixed prosthesis (FPD) etc. Studies have concluded that individuals using CD or RPD show reduced masticatory efficiency as compared to those with natural teeth or FPD. These results are parallel to the present study which found high masticatory efficiency in test group.

Masticatory efficiency is defined as the capacity to grind a certain amount of food in aspecific period of time[4]. Various methods are described to evaluate masticatory efficiency in-vivo. Among these, the use of sieve system is the most commonly used method for measuring efficiency. This method was first introduced by Gaudenz in 1901. Many variations of the the original sieve system are found in literature. It includes the number of the chewing cycle, number of sieve used, diameter of sieve hole and the type of test food used.

In this method, the key point is that, fineness with which the food particles are ground. Quantity of particle reaching the smallest size sieve decide the masticatory efficiency which have direct relation [15].There is a variety of natural test foods which are used in testing masticatory efficiency (like peanut, hazelnut, apple, potato, almond,carrot ham, coconut bread, turnip soya etc) distributed into different classes according to Manley &Bradley and Kapor et al.

According to a study on evaluation of various test foods for measuring masticatory efficiency by Kapoor et al fibrous food like lettuce ,celery were difficult to be chewed, which chestnuts peanuts were the easier one to be chewed and sieved[16,17]. Considering this in the present study peanuts was used as a test food material. Stakes of sieves were used in the present study after considering the study conducted by the Vander Bilt in 2002 where they compared use of single and multiple sieve method for determining masticatory efficiency and recommended multiple sieve method as according to them it provides more accurate results.

Masticatory or bite force is related to the presence of periodontal proprioception.[18]The study found that the mean bite force value of partially edentulous patients were much lesser than the dentate patients, this simulate the result obtained in the present study in terms of bite force. The results described the importance of prosthetic rehabilitation of lost teeth. The mean maximum bite force obtained in the present study on the dentate side was around 305.06N which is near to the values obtained by earlier report[19].

Studies conducted on the comparison of maximum bite force and masticatory efficiency have concluded that there is a direct co-relation between their two parameters. Results of the present study simulate the earlier study. A custom made device to record bite force was used in the present study. Validity of the device, and accuracy with which it can record bite force is needed to be evaluated.

CONCLUSION:

The study result found that there is statistically significant difference in bite force and masticatory efficiency between the flexible RPD and natural dentition . The bite force was more and the amount of material remaing was less in natural dentition side. Therefore it was concluded that the bite force and masticatory efficiency was comparatively less in flexible RPD as compared to the natural dentition.

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A Clinical and Optical Coherence Tomography Study of Coloboma in a Tertiary Health Care Centre of Uttar Pradesh

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Original Research Article

ABSTRACT

This prospective research aimed to study the clinico-etiological features of patients with coloboma, consisted of 124 eyes of 80 patients with coloboma**.** Demographic and clinical data included age at presentation, gender and parental consanguinity. The best-corrected visual acuity was measured with a Snellen chart or Teller chart where possible. The presence of associated ocular anomalies and history of prophylactic laser photocoagulation was recorded. Standard Domain Optical Coherence Tomography (SDOCT) was done in cooperative patients. Mean age was 11.8± 2.25 years (1 month to 25 years). Parental consanguinity was documented in 7(8.75%) of the patients. 44 (55%) patients had bilateral olobomata and 36 (45%) had unilateral involvement. Among 80 patients, 25 (31.25%) cases had anterior colobomas, 31 (38.75%) cases had posterior involvement and 24(30%) cases had both anterior and posterior colobomas. Concurrent ocular anomalies were microphthalmia (32 cases, 40%), amblyopia (24 cases, 30%), strabismus (12 cases, 15%), cataract (10 cases, 12.5%), microcornea (9 cases, 11.25%), and nystagmus (13 cases, 16.25%). OCT in 11 cases showed a "Y-shaped" retina with a fusion of retinal layers and abrupt transition zone between the normal retina into the intercalary membrane (ICM) at the level of the coloboma was seen in 9 cases. Coloboma accounts for 3-11% of blindness in children worldwide. Early onset visual loss have profound consequences on a child's socio-psychological development. Timely diagnosis of coloboma, needful information to the parents regarding the disorder/ anomaly and visual rehabilitation of such subjects should be a priority. Genetic counselling may provide a pivotal role.

KEY WORDS: coloboma, congenital ocular anomalies, optical coherence tomography (OCT)

INTRODUCTION:

The term coloboma is derived from the Greek word koloboma (meaning "mutilated" or "curtailed"). Colobomas are congenital ocular defects (hole in structures of eye) that can affect the iris, the lens, the choroid, the retina, and the optic nerve.[1] Coloboma is defined as a congenital defect in uveal tissue in a site consistent with abnormal closure of the embryonic fissure. Microphthalmos with cyst is a distinct phenotypic form of coloboma.[2] The number of cases is around 0.5 to 0.7 per 10,000 births, making it a relatively rare condition.[3]

The underlying aetiology of the phenotype is the failure of the ectodermal optic vesicle fissure to close.[4] This leads to colobomata affecting one or more areas of the eye including the cornea, iris, ciliary body, lens, retina, choroid, and optic nerve. Eyelid coloboma has also been described, but this is thought to arise from failure of the mesodermal folds to fuse at about 7–8 weeks of gestation. The typical, most commonly observed, ocular coloboma is seen in the inferonasal quadrant.[5] Colobomata in other quadrants are atypical and the embryologic basis for these is unclear. Because the optic fissure forms at the bottom of the eyeball during development, colobomas occur in the lower half of the eye. The particular structure within the eye affected by the coloboma depends on the part of the optic fissure that failed to close.[6]

Ocular colobomata are frequently seen in association with other developmental defects. In the eye, coloboma is often associated with microphthalmos and anophthalmia.[7] Microphthalmos with cyst in one eye may be associated with a coloboma in the fellow eye, and both phenotypes may be seen in different individuals of the same family.[8] People with coloboma may also have cataract,

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glaucoma, myopia, nystagmus), or retinaldetachment.[9] Systemically, a large number of congenital defects are associated with coloboma, including craniofacial anomalies such as cleft lip, skeletal defects such as thumb hypoplasia, and genitourinary anomalies such as horseshoe kidney. Colobomas are seen in Treacher Collins syndrome, in association with cryptophtha-lmos (absence of eyelid formation), Cat eye syndrome (chromosome 22 abnormality with vertical iris colobomas), Patau syndrome (trisomy 13), Fraser syndrome, Manitoba Oculotrichoanal syndrome, Goldenhar syndrome, First arch syndrome, Franceschetti syndrome, Amniotic band syndrome, CHARGE syndrome (composed of coloboma, heart defects, choanal atresia, growth retardation, genital abnormalities and ear abnormalities), renal coloboma syndrome, Aicardi syndrome, Solomon syndrome, and Noonan syndrome.[10]

The grading of the coloboma was done according to the Ida Mann classification[11] as per following details: Type 1: coloboma extending above the anatomic disc; Type 2: coloboma extending up to superior border of disc (Figure 2); Type 3: coloboma extending below the lower border of disc; Type 4: coloboma involving the disc only; Type 5: coloboma present below the disc with normal retina above and below the coloboma (Figure 1); Type 6: pigmentation present in the periphery; Type 7: coloboma involving only the periphery. Media opacities precluding accurate grading were graded as zero for this study.

A complete iris coloboma involves the pigment epithelium and stroma giving rise to the so-called "keyhole" pupil, which can be unilateral or bilateral.[12] A partial coloboma involves only the pupillary margin making the pupil oval. Occasionally, the coloboma only affects the iris pigment epithelium and can be seen only on trans-illumination.[13] Chorioretinal coloboma affecting the posterior segment of the eye can be unilateral or bilateral. If the fetal fissure fails to close posteriorly, then a coloboma affecting the retinal pigment epithelium (RPE), neurosensory retina, or choroid may occur. The defect is a bare sclera with the overlying RPE, retina, or choroid missing. In some cases although the retina is present, it is hypoplastic and gliotic.[12] Typically occurring in the infero-nasal quadrant, it may extend to include the optic nerve. Macular coloboma, which is not due to defects in optic fissure closure, should not be confused with chorioretinal coloboma. The severity varies from no involvement to an obviously enlarged optic cup to gross anomaly unrecognizable as an optic nerve head.[14]

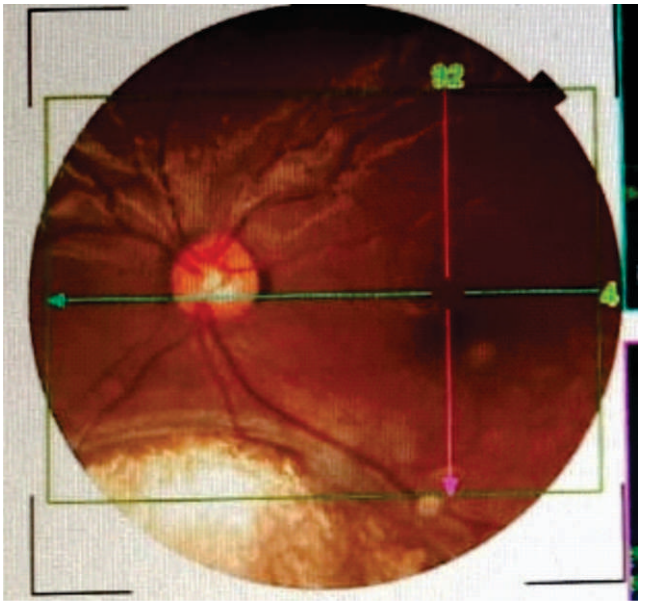

Figure 1: fundus photograph of left eye of a 6 years old male patient showing coloboma present below the disc with normal retina and optic disc above.

Coloboma may occur by genetic, environmental (teratogens),[15] or unknown factors. Autosomal dominant (AD) inheritance is the most common mechanism reported, although most cases are sporadic.[16] Consanguineous pedigrees do support autosomal recessive inheritance for both colobomatous microphthalmos[17] and microphthalmos with cyst.[18] An unusual molecular mechanism, such as trinucleotide expansion, has been suggested for one pedigree with isolated coloboma.[19] Studies suggest that the use of various drugs during pregnancy may be associated with ocular coloboma. Children of expectant mothers treated with thalidomide manifested a number of eye malformations including coloboma (4%) and microphthalmos (7%).[20] Eye abnormalities have been shown to occur in over 90% of children with fetal alcohol syndrome (proportion having coloboma).[21]

Optical coherence tomography (OCT) studies show that the margin of the coloboma either gradual transitions or sudden change into an intercalary membrane (ICM).[22] This ICM can include inner retina, glial tissue, and thin connective tissue.[23] (Figure: 3).

MATERIALS AND METHODS:

A total of 124 eyes of 80 patients having coloboma (inclusive of 44 patients with bilateral findings attending) the Outdoor Patient Department of Maharani Laxmi Bai Medical College, Jhansi, Uttar

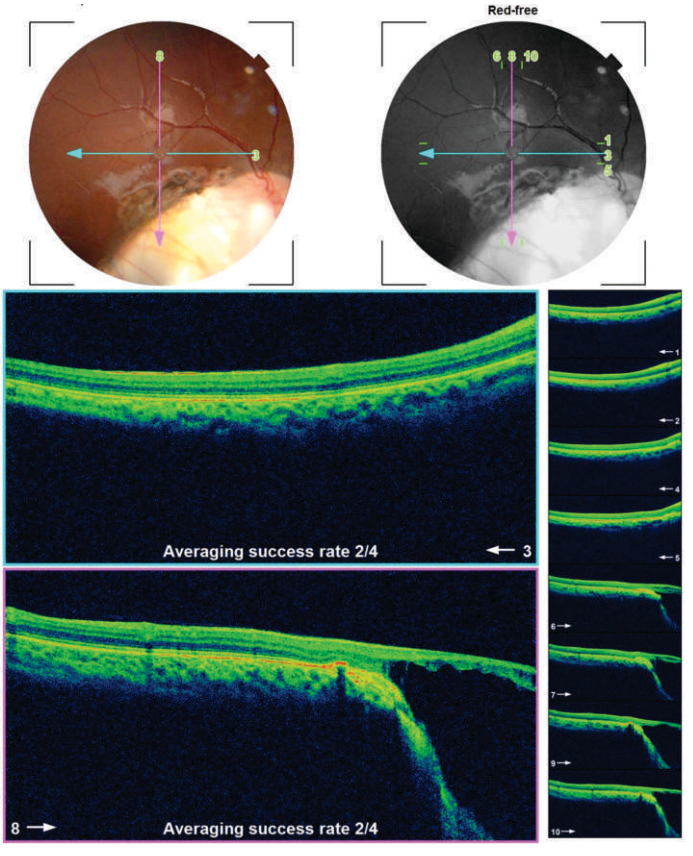

Figure 2: OCT image of right eye of a 17 years old female patient with coloboma extending upto superior border of disc and tomography showing an excavated "Y-shaped" image in coloboma.

Pradesh, India were included in this observational prospective study over a period of 15 months from October 2018 to December 2019. All methods were adhered to the tenets of the ethical standards committee on human experimentation (institutional or regional) and with the Helsinki Declaration of 1975, as revised in 2000. The purpose of this research was to study clinico-etiological features of patients with coloboma.

The inclusion criteria comprised of (a) ≤ 25 years of age; (b) colobomatous malformation in either eye without associated systemic features. Children with non-syndromic ocular anomalies were selected in order to exclude dysmorphic children who were likely to have chromosomal abnormalities or phenocopies due to a teratogen; (c) patients and/or guardians giving proper consent to comply with study requirements. The exclusion criteria comprised of (a) Subjects with clearly identifiable systemic syndrome were excluded; (b) patients having developmental eye abnormalities described as "coloboma" by the referring physician, but who had defects not related to optic fissure closure; and (c) cases with history of laser photocoagulation or any ocular surgical intervention.

Every case was examined by the same ophthalmologist. The anterior segment was examined with slit lamp. Dilated fundal examination was performed by direct and indirect ophthalmoscopy. Each case was examined for systemic abnormalities by the ophthalmologist and a paediatrician/ physician. Demographic and clinical data included age at presentation, gender and parental consanguinity. For infants and developmentally delayed children who could not participate in formal visual acuity assessment, the ability of the patient to fix and follow, the presence of any ocular fixation preference, and/or binocular Teller visual acuity was noted. The best-corrected visual acuity was measured with a Snellen chart or Teller chart where possible. The presence of associated ocular anomalies and history of prophylactic laser photocoagulation was recorded. Standard Domain Optical Coherence Tomography (SDOCT) was done in cooperative patients.

The patient information was recorded in data collection form. Quantitative data were expressed as Mean ± SD (Standard Deviation) and qualitative

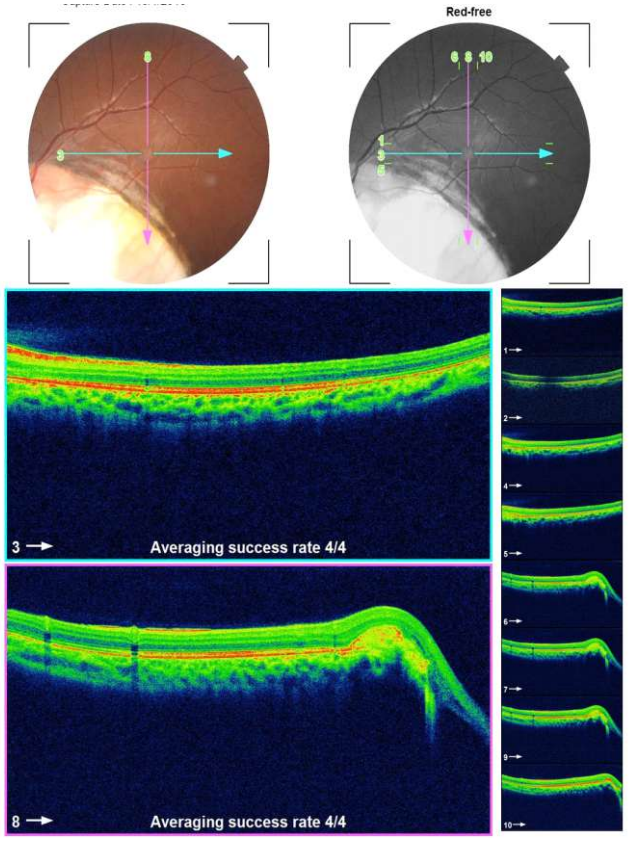

Figure 3: OCT image of left eye of a 21 years old male patient with coloboma extending upto disc and tomography showing that the margin of the coloboma having gradual transitions or sudden change into an intercalary membrane (ICM).

variables were expressed using percentages. Statistical analysis was performed using data analyses tool of Microsoft Excel 2007.

RESULTS:

One hundred and twenty four eyes of 80 patients were studied, comprising 45 (56.25%) males and 35 (43.75%) females. Mean age at presentation was 11.8± 2.25 years (range 1 month to 25 years) . Parental consanguinity was documented in 7 (8.75%) of the patients (Table 1). There were 44 (55%) patients with bilateral colobomata and 36 (45%) with unilateral involvement. Among 80 patients, 25 (31.25%) cases had only anterior colobomas, 31(38.75%) cases had only posterior involvement and 24 (30%) cases had both anterior and posterior colobomas Among the 44 patients with bilateral involvement, 6 had bilateral anterior segment involvement (lens and/or iris) only, 17 had bilateral posterior segment involvement (optic disc and/or retina) only, and 21 had involvement of both the anterior and posterior segments. (Table: 2)

Table 1: Demography of patients in the study.

Age (years)	Mean ± SD
Range (1month -25 years)	11.8 ± 2.25
Gender (Total 80)	Male 45 (56.25%) Female 35 (43.75%)
Parental consanguinity	7 (8.75%)

Visual acuity could not be quantified in 45 patients (56.25%,n=80) because of young age or developmental delay. Among the 45 patients in whom visual acuity could be assessed, 19 patients (42.22%,n=45) had visual acuity (>20/60 in at least 1 eye), 15 patients (33.33%,n=45) were visually impaired (<20/60 but >20/200 in at least 1 eye), and 11 patients (24.44%,n=45) were legally blind (≤20/200 in the better eye). Visual acuity of the eye affected with

Table 2 : Ocular features in cases under study.

Coloboma	unilateral	bilateral	Total (n)
Total cases	36(45%)	44(55%)	80
Total eyes	36	88	124
Anterior segment coloboma	19 (23.75%)	6 (7.5%)	25(31.25%)
Posterior segment coloboma	14 (17.5%)	17(21.25%)	31(38.75%)
Both anterior segment and posterior segment	3(3.75%)	21(26.25%)	24(30%)

Table 3: visual acuity and other ocular anomalies seen in cases.

Visual acuity in at least one eye	Number of patients N=45	Percentage
≥20/60	19	42.22%
≤20/60 to ≥20/200	15	33.33%
≤20/200	11	24.44%
Concurrent ocular anomalies	Anomaly	Total N=80
	Microphthalmia	32(40%)
	Strabismus	12(15%)
	Amblyopia	24(30%)
	Nystagmus	13(16.25%)
	Cataract	10(12.5%)
	Microcornea	9(11.25%)
	Retinal detachment	2(2.5%)

coloboma ranged from 20/40 to no light perception (NLP) All eyes with only anterior segment involvement had vision ≥20/40. Eyes with combined retinal and optic disc involvement, as well as eyes that were microphthalmic, had a higher frequency of visual impairment. Concurrent ocular abnormalities in order of decreasing frequency in total 80 subjects were microphthalmia (32 cases,40%), amblyopia (24 cases,30%), strabismus (12 cases,15%), cataract (10 cases,12.5%), microcornea (9 cases,11.25%), and nystagmus (13 cases,16.25%) **(**Table 3**).**

Standard domain OCT could be performed with successful imaging in 28 cases out of 80. SD-OCT in 11 cases showed a "Y-shaped" retina with a fusion of retinal layers, not being able to identify any retinal layers at this level, toward the edge of the coloboma (Figure 2). The abrupt transition zone between the normal retina into the intercalary membrane (ICM) at the level of the coloboma was seen in 9 cases with chorioretinal layers but lacking the typical layering of normal retina (Figure 3).

DISCUSSION:

A coloboma of the choroid is characterized by congenital absence of part of the retinal pigment epithelium and choroid. It appears clinically as a prominent white zone within the ocular fundus, usually in the inferonasal quadrant. The lesion consists of a rudimentary retina with a few blood vessels over the sclera, which may be ectatic. The neurosensory retina continues as the intercalary membrane (ICM) in the area of the coloboma.[22] Uveal coloboma can exist in isolation or in association with other ocular and systemic abnormalities. In the series described by Mann, the most frequent coloboma extended to and

beyond the superior aspect of the optic nerve and likely involved the papillomacular fibres.[11] In our study, 58 eyes (46.77%) out of 124 included eyes were classified as type 1 or 2 and had vision less than 20/200. Other factors accounting for the reduced vision were lens opacities and associated ocular anomalies, which coexisted in 92 eyes (74.1%). Our study results (Table 2) were in resonance with a retrospective review of Nakamura and associates showing that 36% of patients had only anterior colobomas, 39% had only posterior involvement, and 29% had both anterior and posterior colobomas.[9] Consistent with previous reports, patients with coloboma affecting the retina and optic nerve and those with microphthalmia have a more guarded visual prognosis.[24] Compared to Nakamura's study, we found a lower prevalence of strabismus (15% vs 30%) among our patients. Shah also reported that 44% of their subjects were bilaterally visually impaired (defined as BCVA "outside normal limits") or severely visually impaired (defined as BCVA <3/60). 0ut of 45 patients whose visual acquity could be assessed we found that 33.33% of our patients were visually impaired (<20/60 but >20/200 in at least 1 eye) and 24.44% were legally blind (<20/200 in the better eye) 28.88% of our patients were bilaterally visually impaired or severely visually impaired. Differences in findings among studies may be explained by ascertainment methodology, examination techniques or demographics. A higher proportion of familial cases in India than elsewhere may be related to the prevailing high levels of consanguinity in the population increasing the frequency of recessive disease and modifying the genetic background. However, even in India the majority of cases are sporadic. Possible causes for sporadic cases include unrecognized recessive disease, new mutations, phenocopies (environmental factors), extramarital conceptions, chromosomal aberrations, or complex genetic mechanisms such as polygenic inheritance or gene-environment interactions.[25] Our study found 7 cases out of 80 patients with parental consanguity. This could be attributed to lower consanguity in north India comparing to south India. Optic coherence tomography (OCT) of the optic nerve can help elucidate the different optic nerve anomalies that look similar on fundoscopy alone. OCT of optic nerve coloboma shows retinochoroidal-scleral excavation of the nerve. And OCT of chorioretinal coloboma may depict that neural retina continues as ICM in the area of the coloboma.[22] This transition may be abrupt or, more often, gradual.[26] The outer layers of the retina disappear while inner layers turn into the ICM. Our cases also showed these changes (Figure: 1& 2).

CONCLUSION:

This study delineates the etiological and clinical profile of coloboma emphasizing on proper ophthalmic diagnosis, role of OCT in fundal imaging and visual assessment of patients with coloboma. Such children may be assisted with low vision devices and rehabilitation measures. Childhood blindness has implications for infants' development, education, and future social, marital, and economic prospects. Early onset visual loss can have profound consequences on a child's motor, social, emotional, and psychological development. Coloboma can be isolated, associated with other ocular anomaly and/or associated with systemic manifestation. The visual prognosis in such eyes is linked to severity of ocular malformation and also takes into account the CNS and systemic abnormalities commonly seen with ocular coloboma. Timely diagnosis of coloboma, needful information to the parents regarding the anomaly and visual rehabilitation of such subjects should be a priority. Genetic counselling may also provide a pivotal role.

LIMITATIONS OF THE STUDY:

The limitations of our study are limited number of studied eyes, no follow up study included, difficulties to obtain the images of the entire colobomatous cavities and from the deepest area of the colobomas.

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A Case of Atypical Pituitary Macro Adenoma

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Case Report

ABSTRACT

We report a case of 17 years old male with chief complaints of severe right hemi cranial headache since 3 days for which he seeks advice from local doctor, but did not get relief. Patient also has short height and markedly decreased vision since last 6 weeks. Patient under gone for MRI brain with contrast revealed a large "figure of 8" markedly enhancing sellar mass with suprasellar and right para sellar extension s/o atypical pituitary macro adenoma. Markedly raised Serum prolactin (470ng/ml) and mildly reduced FSH & LH were noted.

KEY WORDS: atypical pituitary macro adenoma, micro-adenomas, panhypopituitarism, pituitary apoplexy, prolactinoma

INTRODUCTION:

The sellar region is an anatomically complex area bounded by sphenoid sinus antero-inferorly, the paired cavernous sinuses laterally, the suprasellar cistern and its contents, diaphragma sellae and hypothalamus superiorly and dorsum sella & brainstem posteriorly.[1]Pituitary tumors are common in the sellar area. The prevalence of clinically apparent pituitary lesions is estimated to comprise approximately 10% of all intracranial lesions.[2] In most cases, they represent slowly growing, clinically non functioning tumours developing from adenohypophysial cells.[3] The mean pituitary volume in the age group 11 to 20 yrs is $0.340cc^3$ in male and $0.280cc^3$ in females.[4] Micro -adenomas are tumors measuring less than 1 cm in diameter and those of more than 1cm are termed macro-adenomas. Diffuse adenomas lead to sellar expansion; often compressing the residual gland into a thin membrane. Massive adenomas often replace the sellar floor; displace surrounding structures and undergo suprasellar extension. Macro-adenomas compress normal pituitary and cause panhypopituitarism. Macro-adenomas often produce stalk effect, in which mild to moderate elevations of prolactin (PRL) hormone result from stalk compression caused by growing tumor mass. This mass blocks the transport of dopamine and thus releases the anterior pituitary from the inhibitory control by the hypothalamus.

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CASE REPORT:

A 17 years old male presented to Medicine OPD with chief complaints of severe headache since 3 days for which he seeks advice from local doctor but did not get relief. Patient also has inability to gain height with increasing age since last 5 years with markedly decreased vision since last 6 weeks. Patient was referred to Department of Radio diagnosis, People's College of Medical Sciences & Research Centre, Bhopal for MRI brain in pituitary protocol to diagnose any pituitary/sellar pathology. The patient was advised for LH, FSH, Serum prolactin, serum cortisol by 8AM/ 4PM and T4/TSH.

The reports were as follows: Serum Prolactin: 470ng/ml (Ref Range 3-35 ng/ml); FSH: 0.99 mlU/ml (Ref Range 1.7-12 Miu/ml); LH: 0.60 mlU/ml (Ref Range 1.1-7 mlU/ml); T4: 71.65 nmol/L (Ref Range 60-160 nmol/l); TSH: 0.638 mircolU/ml (Hypothyroid 0.15-0.25, Euthyroid 0.25-4, Hyperthyroid >5); Serum cortisol morning: 25.3 microg/dl (Ref Range 4.3-22.4); Evening 26.11 microg/dl (Ref Range 3.03-16.66).

In MRI, there is a large well defined markedly enhancing "figure of 8" appearance soft tissue mass measures 4.3 x 2.3 x 2.1 cm (CC x TR x AP axis) in pituitary fossa causing widening of the sella with the suprasellar and right parasellar extension. There were few non enhancing areas within the mass with multiple small areas of blooming in GRE image suggestive of focal haemorrhages. It appears that mass is encasing internal carotid artery at

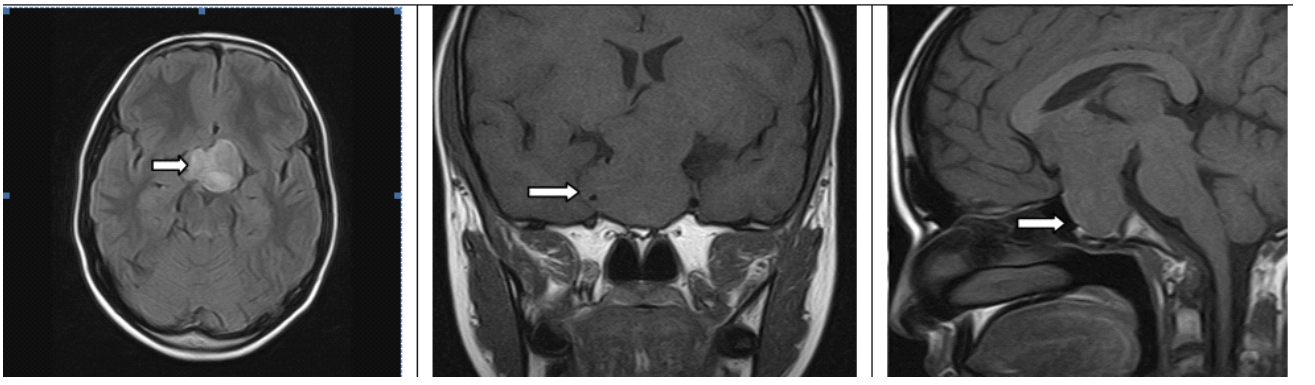


Figure1: Plain, axial, coronal and Sagittal T1 image of brain showing a large heterogeneous soft tissue mass in pituitary fossa causing widening of sella.

its bifurcation along with posterior communicating artery and causing invasion of right cavernous sinus.

Optic chiasm is markedly compressed and displaced postero-superiorily, predominantly left side. In superior extension, mass is extending up to 3rd ventricle with mild indentation with no evidence of hydrocephalous at the time.

Diagnosis of atypical macro adenoma was made on the basis of morphology and change in signal pattern in MRI. Histopathological diagnosis cannot be established because no stereotactic biopsy or surgery was performed. The treatment included Tablet Cabergolin 0.25 mg oral twice a week i.e. Tuesday and Friday and advised for follow up after 3 months. After treatment in Department of Medicine of People's College of Medicine and Research Centre, the patient came for follow up MRI in Department of Radiodiagnosis.

In follow up after 3 months: MRI brain with contrast revealed T2/FLAIR mixed signal intensity soft tissue lesion in pituitary fossa with widening of sella. Few non enhancing necrotic areas and areas of blooming in GRE suggestive of hemorrhage noted within it. It measures 2.4 x 1.7 x 1.5 cm (CC x TR x AP axis) in size. Sagging of optic chiasm with close abutment of bilateral internal carotid artery (right > left) is noted. In comparison to previous scan of before treatment there is marked reduction in size and volume of the lesion with areas of focal necrosis and hemorrhage, significant reduction in mass effect to adjacent structures is also noted. Patient very well responded to clinical symptoms after cabergolin therapy. Visual disturbance and headache relieved completely. Hemorrhage and infarction is shown following cabergolin therapy.

In follow up after 6 months: The current contrast MRI reveal mild reduction in size measuring 1.0x 1.5 x 1.4cm (CC x TR x AP). No obvious mass effect is noted.

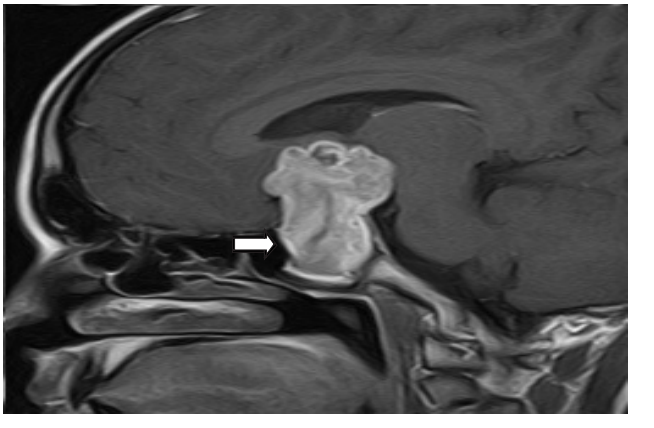


Figure 2: Post contrast TI Sagittal image shows markedly enhancing "figure of 8" appearance soft tissue mass in pituitary fossa causing widening of the sella.

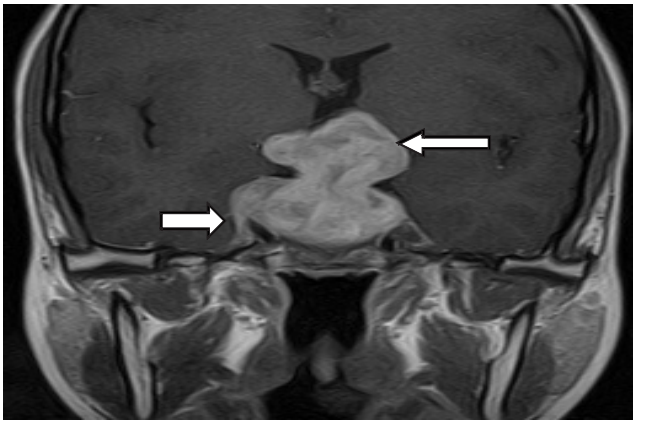


Figure3: Post contrast TI coronal image shows suprasellar and right parasellar extension.

In follow up after 9 months: There is further mild reduction in size of residual pituitary measuring 0.8 x 1.4 x 1.2 Cm (CC x TR x AP axis), with few tiny areas of necrosis noted within it . There is also widening of sella tursica and supra sellar cisterns noted with shagging of optic chiasm. No obvious mass effect or acute hemorrhage is noted within.

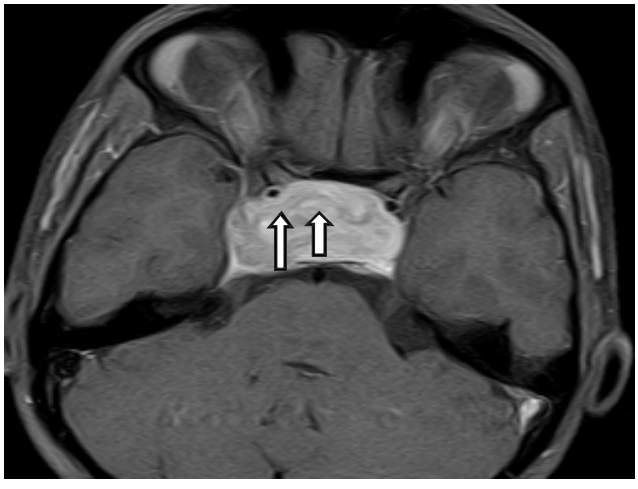


Figure 4: Axial post contrast T1 image shows few non enhancing necrotic areas noted within the mass

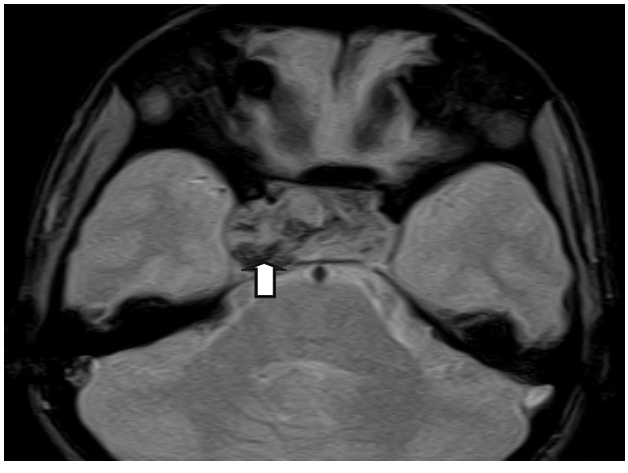


Figure 5: Plain Axial GRE image shows multiple small areas of blooming in GRE suggestive of focal haemorrhages.

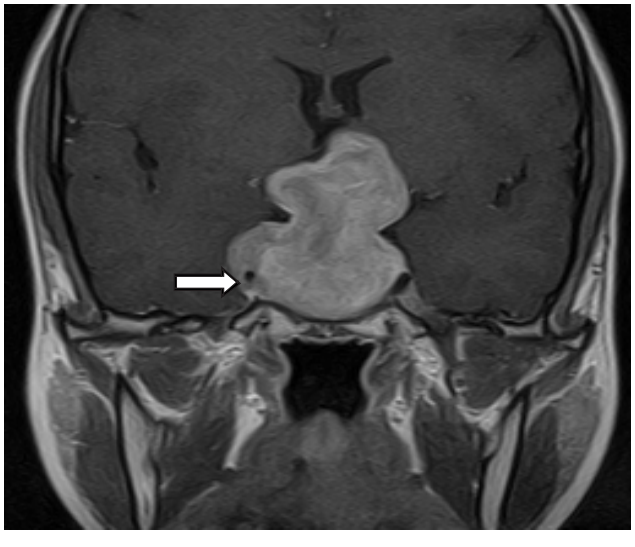


Figure 6: Post contrast TI coronal image shows that mass is encasing internal carotid artery

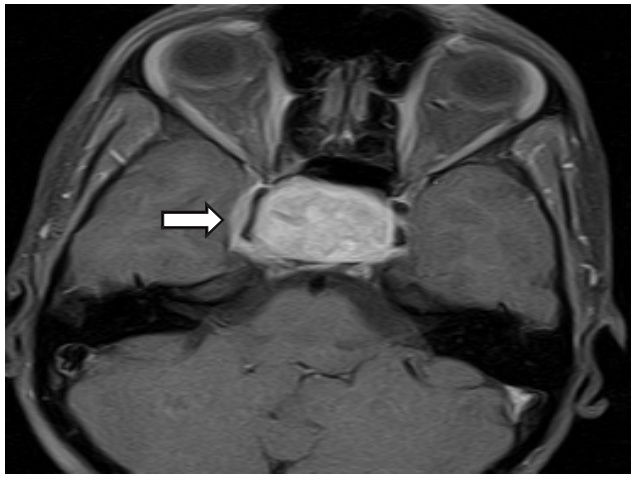


Figure 7: Post contrast TI axial image shows encasement of internal carotid artery at its bifurcation with posterior communicating artery causing invasion of right cavernous sinus.

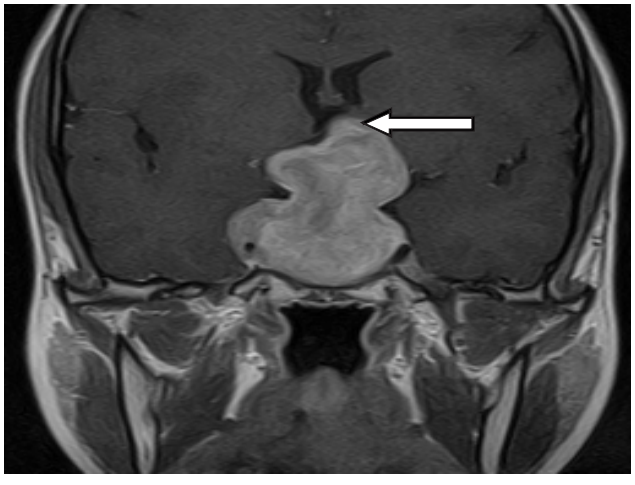


Figure 8: Post contrast TI coronal image shows that Optic chiasm is markedly compressed and displaced postero-superiorily.

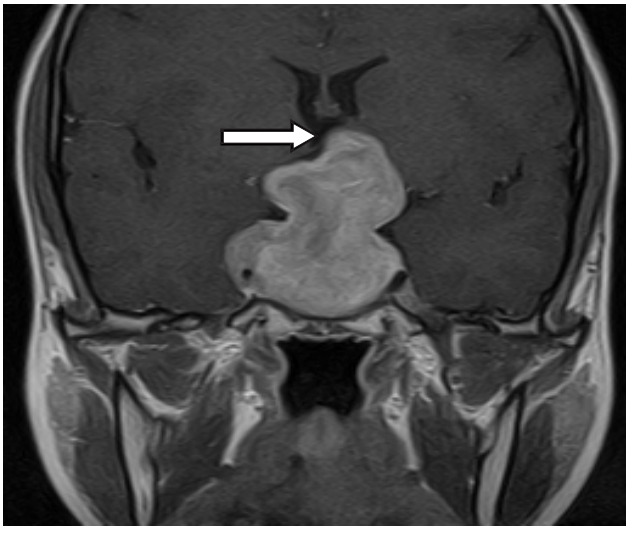
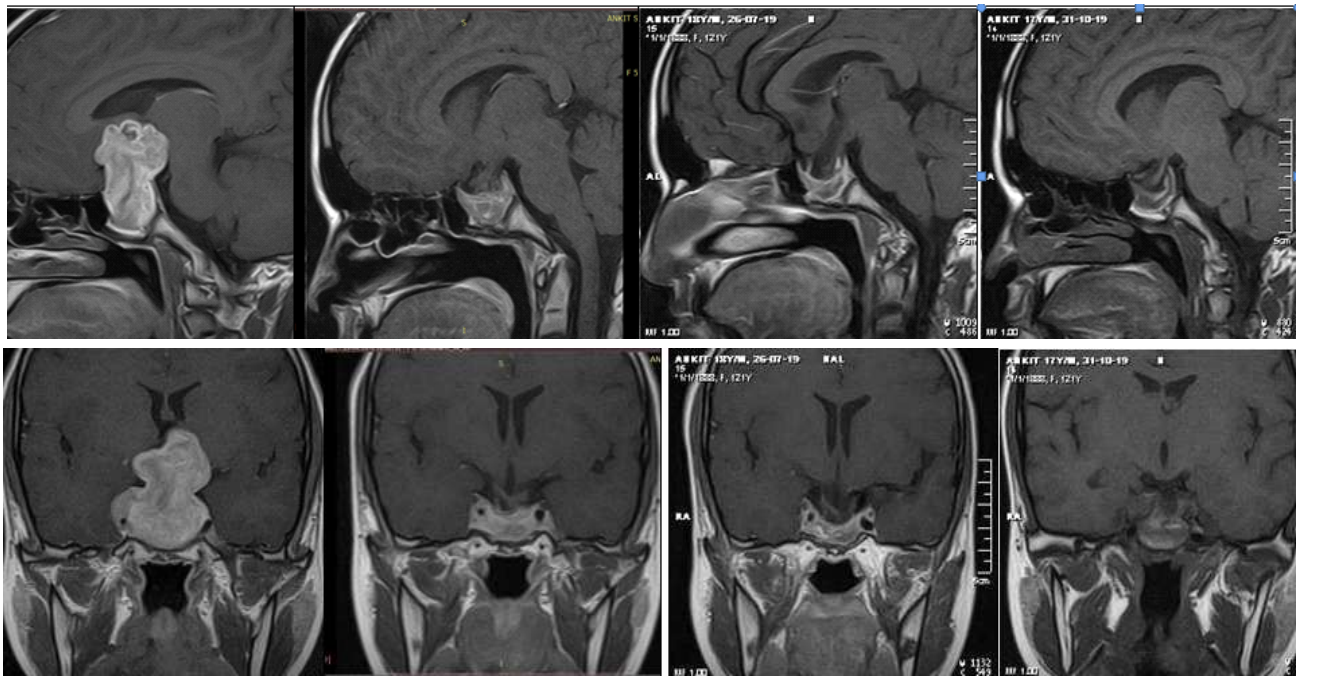


Figure 9: Post contrast TI coronal image shows that mass is extending up to 3rd ventricle with mild indentation.



Before treatment Volume (10.8cc) 3 month after treament (3.2cc) 6 month after treatmetn (1.1cc) 9 month after treamtment (0.7cc)

Figure 10: Treatment response of Pituitary macro adenoma by post contrast MRI in sagittal plane (upper row) and coronal plane (lower row).

The volume of pituitary gland is estimated by using the formula: V= Craniocaudal dimension X Transverse dimension X Antero-posterior dimension X 0.52 (This factor is obtained from the sphere volume equation coefficient and cubic volume calculation: $(4/3\pi)(r_3)/(2r)_3=3.1416/6=0.52$).

DISCUSSION:

Atypical Imaging findings of pituitary adenoma show the following: (a) ectopic location: sphenoid, cavernous sinus, pituitary stalk; (b) abnormal growth / invasion: sphenoid sinus, cavernous sinus, clivus; (c) large haemorrhage, heavily calcified; and, (d) very large size: Giant pituitary adenoma (>4 cm in diameter). Typical Imaging findings of pituitary adenoma show the following: (a) sellar mass with no clear distinction from the pituitary gland; (b) “figure-of-eight " or " Snowman " appearance; (c) size is > 1 (cm); and (d) typically upward growth.

We can only refer to this tumour as an atypical pituitary adenoma, because this tumour presents with aggressive biological behaviour and atypical pattern on MRI brain imaging.

MRI scans showed that the tumour had invaded the surrounding sellar structures, including the cavernous sinus, sphenoid sinus, encasing internal carotid artery at its bifurcation and showing markedly compressed and displaced optic chiasm postero-superiorily, & multiple small areas of blooming in GRE sequence. Early identification of aggressive endocrine tumours would allow the implementation of an intensive treatment that could prevent the recurrence or metastasis. Recurrence rates of 30% for adenomas after a trans cranial approach have been reported .[5] In 2005, Saeger et al. reported an incidence of 2.7% of atypical pituitary tumours among 451 cases from the German Pituitary Tumour Registry who underwent transphenoidal surgery.[6]Atypical pituitary adenoma are thought to be precursor lesions of pituitary carcinoma. Pituitary carcinoma is a very uncommon condition that accounts for merely 0.1% of all pituitary adenomas.

Pharmacological treatment of pituitary macro adenoma is most effective and safest therapy. Previously bromocriptine was used but now-a-days a newer D2 agonist named as cabergolin is used which is more selective for pituitary lactotrope D2 receptors and long acting ($t^{1/2}$ > 60 hours). Just two doses of cabergolin in a week give excellent result with lower incidence of nausea and vomiting. Most of the cases of macro adenoma show significant regression in size and neurological symptoms during therapy of 3

months. Serum prolactin usually returns to normal range in 2-4 weeks of cabergolin therapy.[7]

CONCLUSION:

Abnormal growth and invasion of sphenoid sinus, cavernous sinus, clivus with Large haemorrhage and very large size (>4 cm in diameter) makes the diagnosis of atypical adenomas more reliable. MRI brain with pituitary protocol is the investigation of choice for evaluating pituitary or sellar pathology. It not only helps in the diagnostic differentiation of these lesions but also provides useful information about the anatomical relationship of the gland with adjacent structures and helps to plan surgical approach and also makes a direct comparison with the follow up studies much simpler.

MRI is an essential tool for follow up patients treated clinically by giving cabergoline therapy, as well as in post-operative cases, to evaluate the response to treatment. When young patients are diagnosed with pituitary masses as a part of genetic syndrome then families of young patient should be offered genetic counselling.

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Endodontic Management of Mandibular Biradicular Canine

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Case Report

ABSTRACT

The mandibular canine is placed at the corner of the mouth hence commonly referred to as the cornerstone of the dental arch. It the longest tooth in the mouth usually having a single root and a single canal. The aim of this case report is to highlight the treatment protocol of a mandibular canine with two roots and two canals .

KEY WORDS: anatomical variations, biradicular canine; mandibular biradicual canine

INTRODUCTION:

The mandibular canine is an important tooth in the dental arch. Speculations are that the posterior teeth are protected laterally by the canine guided occlusion because of the anatomic location, anatomy and proprioceptive properties of the canine[1]. Therefore preservation of the canine becomes of prime importance irrespective of its morphologic variation.

The mandibular canine usually is single rooted with a single canal[2]. However, with the advent of newer diagnostic aids and magnification tools the morphological variations of the root canals are perceptible. It is an implicit obligation for the endodontists to have knowledge of the root canal morphology and then initiate root canal therapy to avoid any unforeseen complication.

CASE REPORT:

A 42-year-old female patient reported to the Department of Conservative Dentistry and Endodontics of Mahatma Gandhi Dental College and Hospital, Jaipur for examination of the left mandibular canine (#33) with chief complaint of severe pain in the same. The pain continued for several minutes even after the removal of stimulus, and also leading to disturbed sleep.

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Clinical examination revealed large carious lesions involving the distal aspect and mesial aspect of the mandibular canine and 1st premolar respectively. She had an uneventful medical or allergic history. Radiographic examination revealed sudden loss in continuity of canal in relation to 33 (Figure 1). On the basis of the aforementioned findings, the diagnosis of Irreversible pulpitis in relation to 33, 34 were made. Hence Non-surgical Root canal therapy followed by coronal prosthesis was the binding treatment plan.

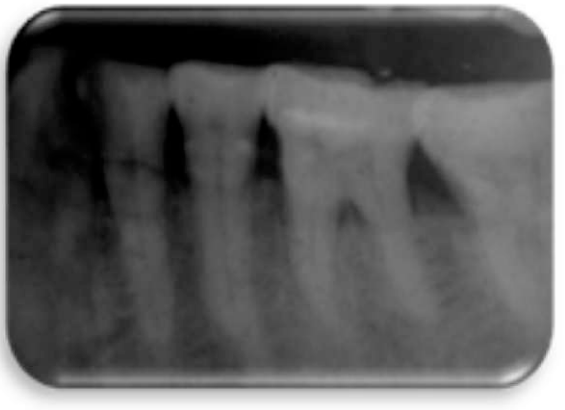

Figure 1: Preoperative IOPA.

Emergency Access cavity was prepared with 33 and 34 under Rubber Dam Isolation after anaesthetizing the tooth using an Endo Access bur (Dentsply Maillefer, Switzerland). Canal orifices were negotiated with DG16 and Working length was determined using a 10 K file (Figure 2). CBCT confirmed the presence of two roots and two canals irt 33 (Figure 3).

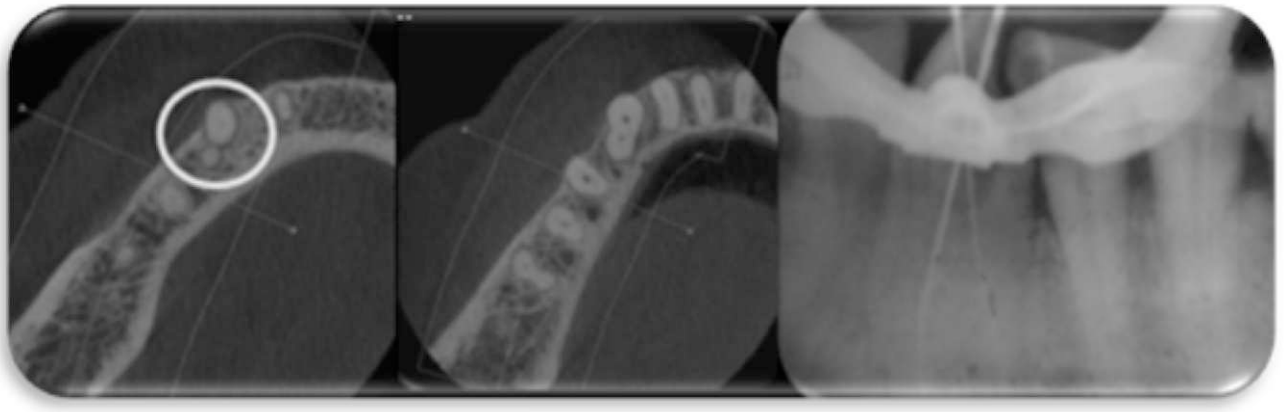

Figure 2 & 3: Working length & CBCT .

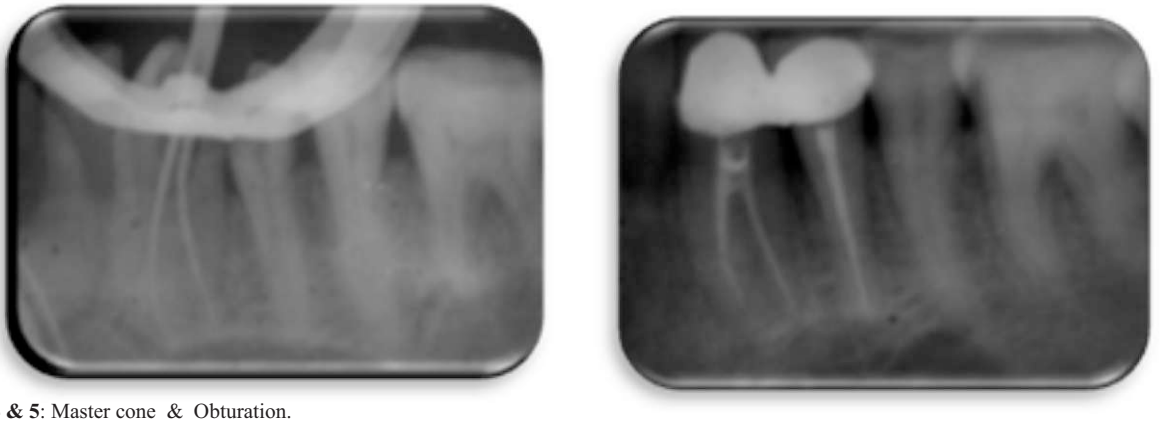

Figure 4 & 5: Master cone & Obturation.

The canal in relation to 33 had a Vertucci Type 4(2-2) and Weine's Type III(2-2) configuration. Biomechanical preparation with 33 was done till F1 in both the buccal and lingual canal using Protaper Gold (Dentsply Maillefer, Switzerland) file system following the Crown Down Technique. Subsequently irrigation was done using 5.2% Sodium Hypochloride, 17% EDTA and Normal Saline followed by calcium hydroxide dressing for 7 days.

In the second appointment, calcium hydroxide removed and canal was irrigated thoroughly with normal saline. Master cone radiograph was taken followed by obturation using the corresponding Guttapercha Cones (Dentsply Maillefer, Switzerland) using a calcium hydroxide based sealer (Sealapex). (Figure 4,5). Tooth 34 was treated as per the standard endodontic protocol.

The patient was recalled after 7 days and crown preparation was done followed by which the prosthesis was delivered.

DISCUSSION:

A thorough knowledge of tooth morphology, careful interpretation, adequate access, and exploration of the tooth are rerequisites for successful root canal treatment[1,2,3].

Canines, morphologically, have a single root[4]. Every mandibular canine are not consistent with a single root and a single canal. There are indications of mandibular canines with unusual findings[5,6].

Having knowledge of internal anatomy relationships is important before taking endodontic therapy for which the periapical radiographs should be carefully evaluated[7]. An additional canal is indicated by sudden change in the radiographic density of the pulp space, or a bifurcation or trifuraction of root is identified by sudden narrowing or disappearance of the root canal space[8].

The Prognosis of Root canal Therapy is governed by the anatomy of the root canal system. One of the major reasons for failure of the treatment is an undetected canal. The treatment can possibly fail from incomplete debridement of the pulp space[9].

The ProTaper Gold was used in this case as its convex triangular cross-section, progressive taper, noncutting tip design allows the instrument to follow the original shape of the root canal. The heat treatment

to the files makes it metallurgically advanced[10]. Also, better root canal cleanliness is obtained by using 17%EDTA which when combined with 5.2% NaOCl provides a greater antimicrobial effect[11].

Good pre-operative radiographs, proper access cavity preparation and optimum obturation are the prerequisites of a successful endodontic treatment. Recent diagnostic aids like CBCT, Dental operating microscope, RVG are the required armamentarium to help complete the diagnosis and plan the appropriate treatment.

CONCLUSION:

Inspite of the low existence of the incidence of mandibular canine with two roots and two canals, they subsit. Root canal peculiarities should be detected using a comprehensive knowledge of the Tooth and Root canal morphology, clinical exploration, intricate radiographic interpretation in addition to the use of Groundbreaking Diagnostic aids.

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Hirayama Disease: Diagnosis to be Missed Without Flexion MRI

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Case Report

ABSTRACT

Hirayama disease, also known as monomelicamyotrophy (MMA), is a rare cervical myelopathy that manifests itself as a self-limited, asymmetrical, slowly progressive atrophic weakness of the forearms and hands predominantly in young males. The forward displacement of the posterior dura of the lower cervical dural canal during neck flexion has been postulated to lead to lower cervical cord atrophy with asymmetric flattening. We report a case of Hirayama disease in a 18-year-old male presenting with gradually progressive asymmetrical weakness and wasting of right hand and forearms.

KEY WORDS: hirayama disease (HD), monomelicamyotrophy (MMA), motor neuron disease (MND)

INTRODUCTION:

Hirayama disease (HD), is a sporadic juvenile muscular atrophy of the distal upper extremities, which predominantly affects the lower cervical cord. It mainly develops in the late teens and early 20's with a male preponderance. The typical clinical features include insidious onset and slow progression of unilateral or bilateral muscular atrophy with weakness of the forearms and hands. Sensory disturbance, autonomic involvement, and upper motor neuron signs like hyperreflexia are rare[1]. The motor neuron disease (MND) is a very close differential diagnosis of HD, but, unlike MND, the disease progresses initially and is followed by spontaneous arrest several years after the onset.

CASE REPORT:

A 18-year-old male presented with a 4 years history of slowly progressive weakness and thenar muscle atrophy that started in the right hand and forearm. The hand weakness limited several activities of his daily living. There was no history of neck pain, sensory involvement, difficulty in walking, bowel or bladder involvement. His past medical history was insignificant; there was no history of trauma, toxins exposure or allergies. None of his family members had a similar complaint.

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Blood investigations were within normal range. Cervical spine MRI was performed on 1.5-tesla siemensmagnetom MR scanner. T1 & T2 weighted spin echo sequences along with diffusion weighted sequence were taken. The MRI in neutral position reveal minimal cord thinning in the region of C4-C5(predominantly involving right half)without obvious cord compression. MRI in flexion position (maximum flexion by patient upto the extent till he don't experience any discomfort), showing loss of dural attachment and anterior dural shift of approximately 4-5mm(extending from C3 to C7 level) with prominent posterior epidural space. The cord is displaced anteriorly. MRI imaging features were consistent with HD.

DISCUSSION:

This disease was initially recognized in Japan in 1959 by Hirayama et al and was reported under the name of "juvenile muscular atrophy of unilateral upper extremity"[2]HD is characterized by insidious onset asymmetrical weakness and wasting of muscles of upper limb with male predominance between 15 & 25 years of age. The disease usually progresses for few years and then is followed by arrest of progression. The clinical features may include irregular coarse tremors in the fingers of the affected hands. The sensory, reflexs, and cranial nerve examinations are generally normal.

Tashiro et al. [3]outlined the criteria for diagnosis of HD: (1) Distal predominant muscle weakness and atrophy in forearm and hand; (2) Involvement of the unilateral upper extremity almost always all the time; (3) Onset between the ages of 10 to early 20s; (4) Insidious onset with gradual progression

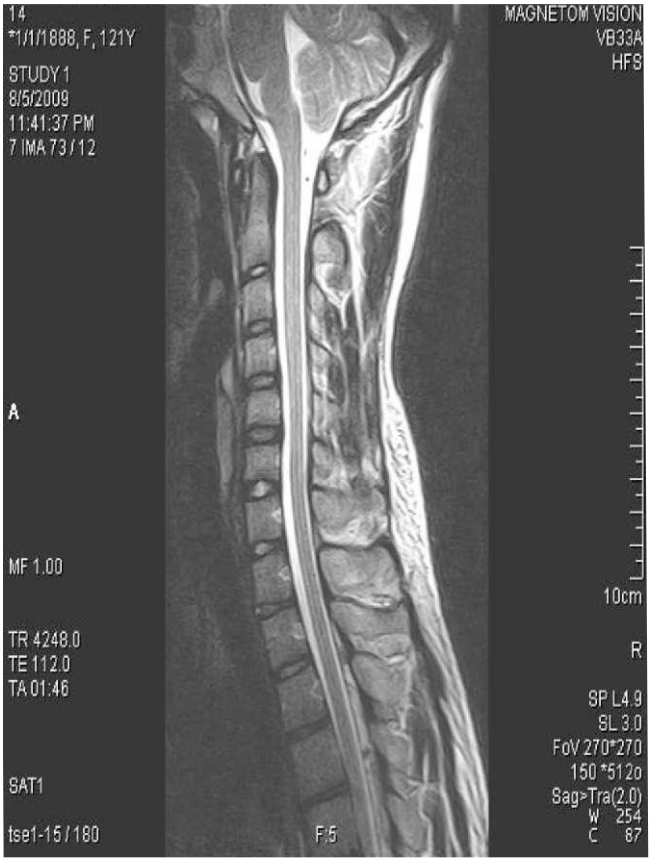

Figure 1: T2 saggital MRI cervical spine in neutral position shows cord thinning at C4-C5 level.

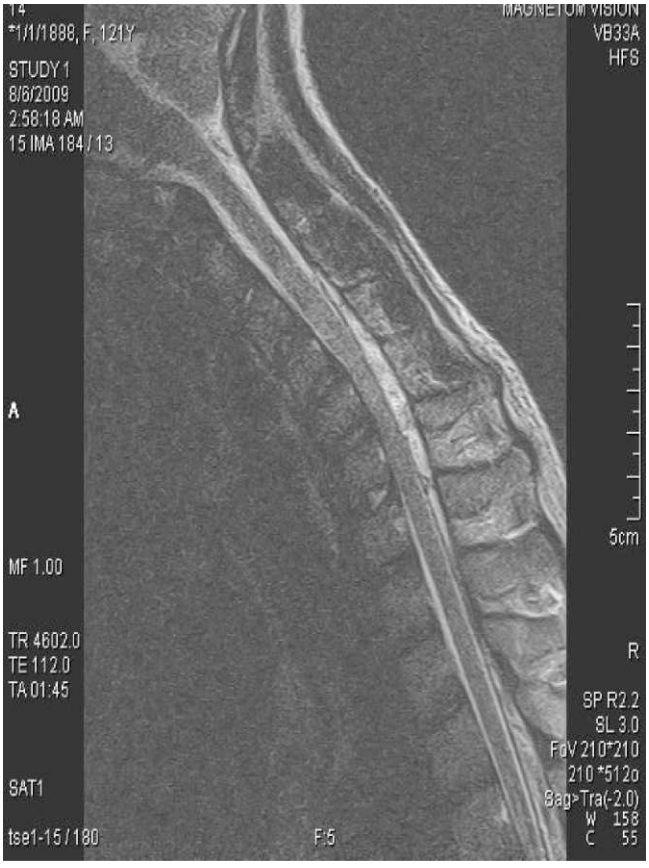

Figure 2: T2 saggital MRI cervical spine in flexion shows anterior dural shift and prominent posterior epidural space.

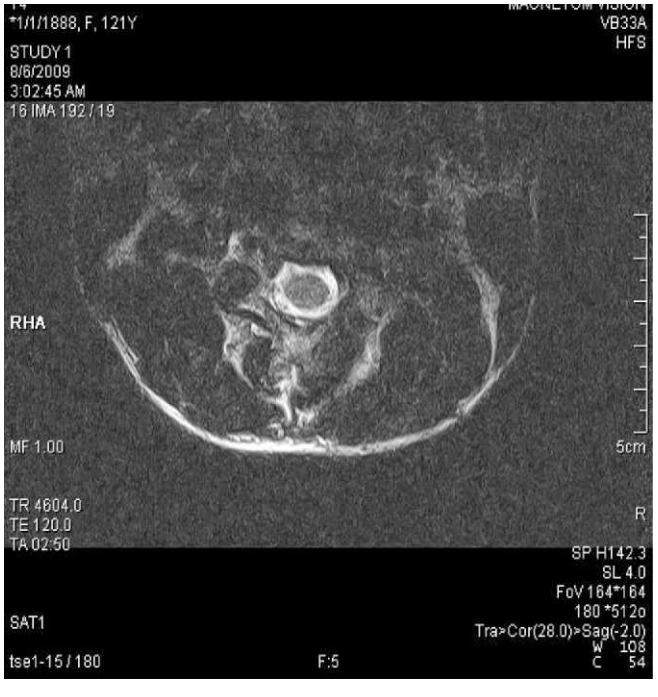

Figure 3: T2 axial MRI shows cord thinning in neutral position.

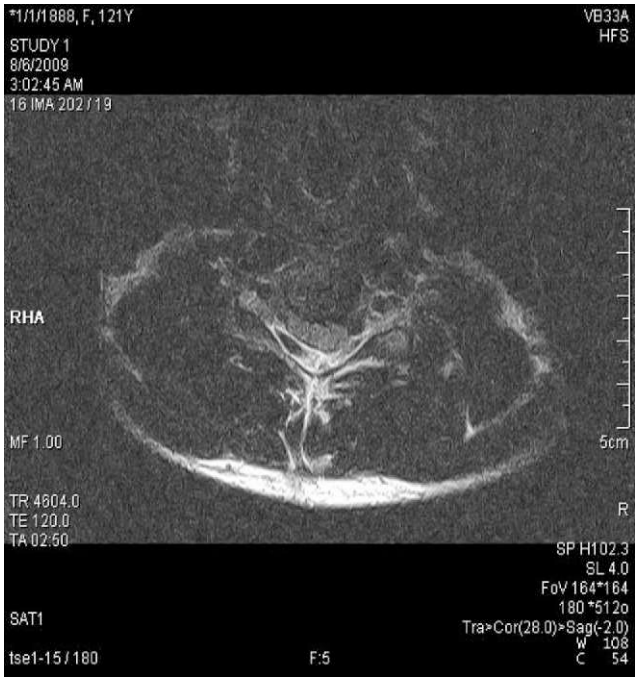

Figure 4: T2 axial MRI shows anterior dural shift in flexion position.

for years, followed by stabilization; (5) No lower extremity involvement; (6) No sensory disturbance and tendon reflex abnormalities; (7) Exclusion of other diseases (e.g., motor neuron disease, multifocal motor neuropathy, brachialplexopathy, syringom-yelia).
The present patient met most of the criteria laid down by Tashiro et al.

The exact pathogenesis of HD is still unknown. A pathological study by Hirayama et al[4] demonstrated cell shrinkage and necrosis, mild gliosis, and some circulatory insufficiency in the anterior horns of the spinal cord from the lower cervical to upper thoracic levels, particularly at the C7 and C8 levels. The most widely accepted hypothesis is a cervical myelopathy associated with neck flexion, proposed by Kikuchi et al[5]. Normally, the spinal dura mater is loosely attached to the vertebral canal by the nerve roots and the periosteum at the foramen magnum and the dorsal surfaces of C2 and C3 and the other at the coccyx. The relatively short and tight dura mater seen in patients with HD is unable to compensate for the increased length of the vertebral canal during neck flexion. This results in tightening of the dural canal during neck flexion, which leads to an anterior shift of the posterior dural wall, causing spinal cord compression against the vertebral body. This repeated neck flexion results in multiple episodes of ischaemia and chronic trauma to the spinal cord, which eventually leads to myelopathy, as evidenced by asymmetric lower cervical cord thinning in the MRI.

The differential diagnosis of HD includes the distal form of spinal muscular atrophy, amyotrophic lateral sclerosis (ALS), multifocal motor neuropathy with conduction block, and toxic neuropathy as well as structural lesions of the cervical cord. The key to diagnose this disease is based on the typical clinical features and dynamic MRI study when the neck is flexed. MR studies in flexion show not only the anterior displacement of the posterior wall but also a well-enhanced crescent-shaped lesion in the posterior epidural space of the lower cervical canal. This lesion typically disappears when the neck returns to a neutral position, confirming it to be a congested posterior internal vertebral venous plexus rather than a vascular malformation.

MR imaging studies of the cervical spine in a neutral position can reveal several features such as localised lower cervical cord atrophy, asymmetrical cord flattening, and loss of attachment between the posterior dural sac and subjacent lamina, as well as noncompressed intramedullary high T2 signal intensity.

CONCLUSION:

Hirayama disease is a self-limiting disorder and there is no consensus on the definitive treatment. However, early diagnosis is necessary because a cervical collar and physiotherapy may arrest the progression of the disorder by limiting the neck flexion.

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